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OF THE

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, October 11, 1904.

DR. J. H. JOPSON, PRESIDENT.

DR. GEORGE B. WOOD showed a patient with

STENOSIS OF THE LARYNX FOLLOWING TYPHOID FEVER.

DR. JOPSON said that he had not seen the child before she was operated upon. In August, he saw the patient with Dr. Wood, and introduced, with considerable difficulty, an intubation tube several sizes smaller than the larynx of a child of that age should accommodate. The patient afterward breathed through the tube, showing that there was no obstruction below it. It was thought best not to leave the tube in, but to attempt gradual dilatation and afterward try to get rid of the tracheotomy tube. A week later, it was found that not even the smallest sized intubation tube could be introduced into the larynx without administering an anesthetic, so its introduction was deferred until an anesthetic could be given. He said that if there should be a large quantity of granulation tissue and an active process of ulceration in the larynx, intubation would not be indicated. In cases of diphtheritic stenosis gradual dilatation by means of intubation tubes has been found better than thyroidotomy; but in this case the space between the cords is filled up with granulation tissue, and the pathologic condition is different from that coming on after diphtheritic involvement of the laryngeal mucous membrane. He agreed with Dr. Wood that it is a question whether these granulations will disappear spontaneously.

DR. M. H. FUSSELL exhibited

A PHOTOGRAPH ILLUSTRATING DR. WOOD'S CASE.

He said that he had seen 2 cases of perichondritis in typhoid fever, one occurring in a boy of seventeen, and the other in a man of thirty. This latter was convalescent from the fever, had only a simple hoarseness in the beginning, and the case was not recognized as serious until the occurrence of a severe cyanosis and stenosis. He refused operation, and died shortly afterward. The picture exhibited showed the result of the autopsy, in which com-

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plete necrosis of the cricoid cartilage was found. The boy of seventeen went through a severe attack of typhoid, and on the fortieth day developed a simple hoarseness. As Dr. Fussell had but shortly before seen the other case, this hoarseness greatly alarmed him. Even although watched carefully, the patient almost died, on account of the sudden final onset of the stenosis, and a delay in the arrival of a surgeon. He was tracheotomized by Dr. A. C. Wood, recovered entirely from the typhoid, was able to withdraw the tube at the end of six months, and lived comfortably afterward with a little stenosis. He was able to attend to his business as before, but about a year ago he died of appendicitis.

According to the statistics in Dr. Keen's book on the "Surgical Complications of Typhoid Fever," Dr. Wood's case is one of the rarest in point of its early age. Only 12 out of 221 reported cases were under fifteen years of age. The statistics also show a great preponderance of males. The laryngitis almost always comes on late in the disease, as a simple hoarseness frequently after convalescence is well established, and is frequently overlooked entirely, until the severe stenosis occurs. One should be on the lookout for this beginning hoarseness, and be prepared to do a tracheotomy upon the onset of stenosis. Dr. Keen's statistics show the mortality to be still very high, probably because in these cases operation was delayed too long. His younger case almost lost his life on account of the delay in the arrival of the surgeon.

Dr. Thompson S. Westcott reported a case with

PECULIAR PRESSURE SYMPTOMS DUE TO ENLARGED TUBERCULOUS BRONCHIAL LYMPH NODES

which occurred in an infant that died at the age of four months. The most advanced lesion was found in one of the bronchial lymph nodes, which was enlarged to the size of a pigeon's egg, and had undergone complete softening. It was so situated as to press upon the descending vena cava. In the last two weeks of life marked cyanosis of the head, neck and upper extremities was produced whenever the baby was held in a position increasing the concavity of the dorsal segment of the spinal column, but disappeared when the child lay flat upon its back. The lungs were studded with miliary tubercles, and one or two were found in the spleen. Death had followed a bronchopneumonia of the right upper lobe.

The child had been born in a private room in one of the city

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hospitals, and had been breast-fed exclusively, never having received artificial food of any kind. The mother and father were in good health, but there was an uncertain history of tuberculosis in the mother's family. The first symptoms of failing health had been noticed at the age of two months. Interest attaches to the case from the fact that at the time the baby was born a patient suffering with advanced pulmonary tuberculosis had occupied another private room across the hall from hers. The early appearance of the first signs of illness (at two months of age), and the advanced stage of disease in the bronchial nodes make it certain that the infection had been received very soon after birth, since the mother was then, and still continues, in good health, and the baby thrived normally until about two months of age, which would practically rule out an intrauterine origin of the disease. In the absence of other known exposure, he was inclined to attribute the primary infective source to the tuberculous patient in the room near the lying-in room at the hospital. He suggested either air-borne infection or more direct conveyance through the medium of nurses, who, in all likelihood, took care of both the mother and infant and the case of tuberculosis.

Dr. Willson said that the marked cyanosis that occurred when the baby was in the erect posture and its disappearance when lying down is contrary to the usual phenomenon in adults when there is tumor of the bronchial nodes pressing upon the trachea or vena cava. In nearly all mediastinal tumors the cyanosis appears or becomes more marked when the patient is in the recumbent posture. He then asked Dr. Westcott whether it had occurred to him that this might be a case of intrauterine tuberculous infection. Dr. Westcott had not stated why he had excluded that diagnosis. He (Dr. Willson) thought it especially probable in this instance, on account of the fact that the child's grandparents had had tuberculosis; that a brother or a sister had rickets; and that another child of the same parents had had a chronic and possibly tubercular diarrhea. An infection in the child itself had admittedly occurred within the first two weeks, it seemed probable to him that it had existed before birth.

Dr. D. J. Milton Miller asked whether any of the ordinary physical signs of enlarged bronchial lymph nodes had been present in this case, viz., dullness under the sternum, paroxysmal or brazen cough, Eustace Smith's sign, etc.

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Dr. Westcott said that there had been no dulness behind the manubrium; no cough until within a few days of death, when bronchopneumonia developed; and no murmur when the head was thrown back.

Dr. Miller then said that he had asked this question because these signs are given in most text books as evidence of the presence of enlarged bronchial lymph nodes; but he had found them very seldom present, for it is not often that the bronchial lymph nodes become sufficiently enlarged to produce signs of pressure such as are caused by aneurysms or mediastinal tumors. Dr. Miller has used the Eustace Smith sign in examining children thought to have enlarged bronchial lymph nodes, but had seldom found it present; neither had he found the paroxysmal cough, resembling pertussis, nor the manubrial dulness nearly so often as is usually stated. In regard to the question of infection, he thought it probable that Dr. Westcott's case might have become infected in the way described by him. He had seen a child that died of acute miliary tuberculosis when quite young, who had lived for the first few months of its life in the same room with its tuberculous father. This man was in the habit of expectorating on the floor, and Dr. Miller thought that the infection had taken place directly through the respiratory passages, as it probably did in Dr. Westcott's patient.

Dr. Weber, who had been present at the autopsy, said that the lymph nodes were attached to the vena cava, and were slightly behind and to the right side. It was easy to see how the upright position with the thorax flexed and the head bent forward would cause the node to press on the vena cava and produce the symptoms.

Dr. Westcott said that he might have laid too much stress upon the family history. There was some tuberculous tendency in the mother's family, and the father seemed to be rather anxious to blame the trouble on the mother and her relatives. The question of tuberculosis was discussed at the time of the first child's death. This had suggested to the father the possibility that the diarrhea in the second child might be tuberculous; but, as it showed no signs of wasting disease, Dr. Westcott combatted that idea. The diarrhea was undoubtedly caused by a subacute catarrh of the bowel, and the child afterward became entirely well. The mother of the child is apparently in perfect health and

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has no evidence of tuberculous disease, and for this reason he thought it somewhat gratuitous to suggest the possibility of intra-uterine infection. The child remained well until the second month, when it began to show signs of failing health. On this fact he had based his belief that the disease had been contracted prior to the second month; and the advanced stage of disorganization of the lymph node indicated that it might well have begun even before that time, probably during the first month; so that it is quite likely that it occurred at the hospital during the first two weeks of the child's life. He said that this case shows that one cannot be too careful about the surroundings of a lying-in patient, and that a general hospital, in which cases of all sorts are received, even in the private wards, is not a safe place to which to send a maternity case.

DR. ALFRED HAND, JR., reported the following cases:

(1) UREMIC ONSET OF TYPHOID IN A CHILD THREE YEARS OLD; RELIEF BY LUMBAR PUNCTURE. (2) URTICARIA AS A SEQUEL OF DIPHTHERIA WHEN ANTITOXIN WAS NOT USED.
(3) UNUSUAL COURSE OF MEASLES RASH.

Full reports of these cases are to be found on page 3 of this volume among the "Papers."

DR. E. E. GRAHAM said that the first case reported by Dr. Hand had interested him greatly, because it was a study of an unusual complication of typhoid fever. Albuminuria, if by this one means a trace of albumin in the urine, is quite a common complication, but acute parenchymatous nephritis is quite rare. Another point of interest is that if such patients recover from the typhoid, they almost invariably recover from the acute nephritis, although the mortality in the cases of acute nephritis complicating typhoid fever is very high. The acute nephritic condition seems to be entirely dependent upon the typhoid toxemia. Dr. Graham said that he judged from the chart that Dr. Hand's patient had recovered from the acute symptoms of uremia, but he did not remember whether there was entire recovery from the nephritis.

DR. HAND, in closing, said that the last urinalysis before the child was discharged was entirely negative for any signs of nephritis.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, November 8, 1904.

JOHN H. JOPSON, M.D., PRESIDENT.

DR. GEO. W. NORRIS showed a patient with

CONGENITAL HEART DISEASE.

Both of the patient's parents have had pulmonary tuberculosis for a number of years, and are now under treatment. The patient, J. C., aged five years, was born a blue baby, and remained so for a short time after birth. He had varicella and pertussis uneventfully, two years ago. When two years old he had an attack of spasmodic croup. At this time he was very ill, and was noticed to be markedly cyanosed. He has been troubled with cough for a number of years. This symptom is always worse in the morning and after exercise, but is unaccompanied by expectoration. He becomes slightly cyanotic upon exertion, also somewhat dyspneic; and the mother says that he is always cold.

The child is somewhat pale in color, but is fairly well nourished. He is of fair complexion, has rather a light frame, but seems, both mentally and physically, developed commensurately with his age.

On physical examination the apex beat of the heart is found in the fifth interspace a trifle to the left of the normal. A fine systolic thrill is felt over the entire precordium. The heart dullness seems to be about normal on the left, but on the right extends fully one inch and a half beyond the edge of the sternum. Between the third and fourth right costal cartilages a loud, long, rasping, high-pitched murmur is heard; which completely replaces the first sound of the heart. It is transmitted with greatest intensity toward the epigastrium, but may be heard at any part of either chest, either anteriorly or posteriorly. The pulmonary second sound does not seem to be accentuated, and the aortic sound is barely audible. The radial pulses are synchronous, equal, regular and of fair volume.

The lungs show an area of impaired resonance posteriorly in the region of the heart. Elsewhere the resonance seems good.

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At no point in the chest is there any alteration in the character of the expiration. A few fine moist crackling râles are heard diffusely scattered throughout both lungs. There is no enlargement of the liver, or other physical abnormality. The pulse is 120, the respiration 40, and the temperature 97° F.

From the character and location of the murmur, as well as from the other physical signs and from the apparent absence of an etiological factor it was concluded that the case is one of congenital heart disease. From the evidence in hand it seems likely that the lesion consists either in a defective ventricular septum, or a patulous foramen ovale, or perhaps both; either with or without a patulous ductus arteriosus. The possibility of tricuspid insufficiency must be considered, although its presence would seem unlikely in the absence of symptoms. It may be stated, however, that no less an authority than Gibson believes that this condition is much more common than is generally supposed; although it is usually overlooked on account of being unproductive of symptoms.

Dr. Hamill said he had never heard a murmur in the peculiar situation of this murmur and with such marked transmission to the right. He thought the possibility of a tricuspid lesion should be considered, though, on the whole, Dr. Norris' explanation of the signs was perhaps the most probable.

Dr. Norris said he had considered the possibility of a tricuspid lesion particularly because at the time of the first examination the murmur was heard best at the tricuspid area. Upon re-examination, however, he became convinced that the maximum intensity was on the sternum at the junction of the third or fourth rib. In addition to this there had been no signs of the mechanical effect of tricuspid regurgitation, such as pulsation of the liver, and he had, therefore, become convinced that the condition was not a tricuspid one.

Drs. C. Y. White and Verner Nisbet showed specimens of a

HYPERNEPHROMA OF THE RIGHT SUPRARENAL GLAND WITH METASTASIS, AND HYPERTROPHY OF THE EXTERNAL GENITALIA IN A CHILD OF TWO YEARS.

The patient from whom the following specimens were obtained was shown before the Philadelphia Pediatric Society on

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October 13, 1903, by Dr. D. J. Milton Miller, as "A Case of Abdominal Tumor (Sarcoma of Kidney?) and Pseudohermaphroditism in a Child of Two Years." (ARCHIVES OF PEDIATRICS, Vol. XX., 1903, p. 933.)

The subsequent history of the case shows a rapid increase of the previously described symptoms—increasing dyspnea, marked congestion of the face, profuse perspiration, luxuriant growth of pubic hair, and great abdominal enlargement. On October 23, 1903, an exploratory laparotomy was performed, but the tumor was found to be so large, adherent and vascular, that removal was considered inadvisable. The child survived the operation, and also an attack of measles and bronchopneumonia, but finally died from exhaustion on September 24, 1904. Toward the end of the disease the general appearance of the child was striking. The dark, but not bronzed skin, anxious, prominent eyes, thick neck, luxuriant pubic hair, hypertrophied genitalia, and short, thick figure (height, 36 inches, abdominal circumference, 24 inches), combined with her waddling gait, gave the appearance of a little elderly woman.

The autopsy was performed twelve hours after death and a large tumor of the right suprarenal gland was found with metastasis to the liver, mesenteric lymph nodes and right lung. The microscopic examination showed the tumor to be a hypernephroma.

The external genitalia were moderately hypertrophied. The pubic region was covered with coarse hair, but no excess of hair was observed on the remainder of the body. The labia were hypertrophied and the labia minora projected from the vulva. The clitoris measured 4 cm. in length and the prepuce could be retracted. The urethral orifice was situated in the vestibule in its normal position. The ovaries were enlarged, but otherwise the internal genitalia were those of a normal female child. The microscopic examination of the tumor will be reported in detail later.

DR. D. J. MILTON MILLER referred to the fact that he had exhibited the child last year before the Society, and that the diagnosis at that time was sarcoma of the kidney with secondary deposits in the liver. A correct diagnosis during life would have been impossible, and, indeed, the origin of the tumor was determined only by a very careful pathological examination. As

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to the nature of the growth, Dr. White, before the microscopic examination, had regarded it as sarcomatous.

Dr. Jopson said that he had seen the case many times during life, and with others had been very much interested in it because of the presence of so large a mass together with maintenance of relatively good health. Dr. Hutchinson had done an exploratory operation, but had found so many adhesions that it was impossible to do anything but close the wound at once. The operation had had no bad effect and the child lived over a year afterward. The relation to precocious puberty is an interesting point and in this condition it has been repeatedly noted. The same condition has been seen also in pelvic tumors in children, and in the latter instance it has been attributed to an increased blood supply. In this case the only evidences of precocious puberty were the precocious growth of pubic hair and the marked development of the clitoris.

Dr. Nisbet, in closing, said it was interesting to know that neither during life nor at autopsy was any bronzing of the skin found.

Dr. D. L. Edsall read a paper on

TYPHOIDAL INSANITY IN CHILDHOOD, WITH A DISCUSSION OF ITS CHARACTERS AND ITS PROGNOSIS.

A study of the literature readily showed over 80 cases and no attempt was made to make the collection of cases complete. Dr. Edsall considered that the condition is undoubtedly somewhat more frequent than is commonly thought, particularly in the milder forms. Mania is unquestionably the commonest form; dementia next most frequent; the next most common group simple delusions, hallucinations, etc.; after these follows simple melancholia without any maniacal tendencies. The prognosis is certainly somewhat more grave than the statements of general clinicians and pediatrists would indicate; though the patients usually get well, a certain number of the manias and melancholias do not recover, and of the dementias reported in the literature, a very large proportion persist. The character of the insanity is, therefore, of great importance in establishing a prognosis. The importance of the nutritional element in these cases was especially insisted upon. Two personal cases were reported —one a simple dementia that recovered; the other, a case of melancholia, with occasional maniacal outbreaks.

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Dr. J. P. Crozer Griffith said he believed with Dr. Edsall that the statistics that had been read showed probably too unfavorable a prognosis, and that the condition is also more frequent than they would indicate. An analogous series of statistics, that emphasized this statement, was the collection of cases of typhoid fever in infants made by Dr. Griffith and Dr. Ostheimer. Typhoid fever in early life is certainly more common than even those statistics would indicate, and certainly has a much smaller mortality than they show, the reason being that the mild cases are either overlooked or, at any rate, never reported, while a relatively large proportion of the severe and fatal cases are reported. The same is undoubtedly true of posttyphoidal insanity in childhood. Of this condition, Dr. Griffith has seen 2 or 3 cases; one that was reported by him some years ago showed conditions much like those in Dr. Edsall's case, *i. e.*, various curious delusions which were usually associated with fear, and also a varying condition of profound depression and excitement.

Another child, a boy eight or ten years old, was of interest because it was so hard to say when the condition began with him. In this case the nutritional element suggested by Dr. Edsall was very evident for the child had been very delirious, he had eaten extremely little, and there had been no attempt to give him the proper amount of food or to stimulate him. The result was that the delirium grew wilder and wilder; and as the fever grew less the delirium persisted and grew worse. For days the boy almost constantly screamed so loudly that he could be heard a long distance from the house; the screams ceasing only when his voice gave out. He gradually improved and ultimately recovered entirely. A considerable number of cases of typhoid fever show much more marked mental confusion than is normal with this disease, and, as Dr. Edsall said, we sometimes see distinct delusions during the course of the disease. As such conditions are sufficient to suggest the possibility that an actual insanity may develop, he agreed that such cases should be fed freely and other measures used in the attempt to prevent any further mental derangement. Usually it does not develop; occasionally, however, it, of course, does, and proper prophylaxis is very important.

Dr. Griffith asked what Dr. Edsall's searches of the literature showed as to the period of development of the insanity; this would have a large influence upon the question whether it is toxic or nutritional. If it appears during the course of the disease, it

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is certainly often toxic. When it appears during convalescence it is probably most commonly nutritional.

Dr. Hamill said that he had learned an early lesson concerning the relation of typhoidal mental derangements to imperfect nutrition. In a case that he had seen when he was Dr. Henry's resident in the Philadelphia Hospital, a liquid diet had been continued because of the persistence of a slightly elevated temperature. The patient constantly demanded food. He became maniacal after a short time, but still persisted in his demand for food. When his diet was increased the mania rapidly disappeared.

Dr. Hamill had never seen actual insanity following typhoid fever in a child, but he had seen numerous cases of marked mental disturbance in children in the course of the disease, and last winter in his service at the Polyclinic Hospital such conditions were so extremely frequent as to be the subject of repeated remark. He meant by this a very excessive irritability in the children and frequent hallucinations or delusions. He mentioned one instance, that of a colored child, that never spoke without yelling at the top of its voice. The child also had hallucinations and delusions. This abnormal state terminated with great abruptness, the child one day suddenly seeming entirely rational.

He believed that these conditions were more common last winter than he had ever seen them before; a fact which would, to some extent, bear out the possibility mentioned by Dr. Edsall, that mental derangements are more common in some epidemics of typhoid fever than in others.

Dr. Eleanor C. Jones described a case she had seen a few months ago in a girl, in which there was very pronounced delirium during the course of the disease, and the delirium continued during convalescence. In this case there were also other symptoms much like those in Dr. Edsall's case. In this instance, the condition was certainly largely nutritional, for when the child's food was quickly increased, it rapidly responded with improvement, and after three weeks was dismissed well.

Dr. Edsall said that he did not wish to be understood as stating that posttyphoidal insanity is always a nutritional condition. It is certainly sometimes toxic, but in a large proportion of cases it is almost, or perhaps wholly, nutritional in its origin; and even when it has almost undoubtedly originated in the toxemia, it persists because of the disturbance of nutrition

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that has resulted from the disease. One has frequent opportunity to be convinced of this in a review of the literature and occasionally in one's own experience. He mentioned a case which was seen a few months before in, which there had been hemorrhages in an adult. The repeated and severe hemorrhages had led to the very marked reduction in the man's diet and necessarily only small amounts of food were given for quite a long time. The man had first begged for more food, but soon he fell into a condition of such marked inanition that he could hardly be forced to take food, and emaciated and lost strength rapidly, and soon became insane. Forced feeding, partly through the stomach tube, produced improvement in this man; but he suddenly died, probably largely as the result of a heart lesion which he had before his typhoid fever came on. The nutritional element is recognized by most alienists as being an exceedingly important point in most cases, particularly in the treatment of the condition. It is certainly an extremely important matter to keep in mind because it is one that is usually so readily controlled.

Dr. Griffith asked whether Dr. Edsall had determined that there was any relation between insanity and aphasia. A number of authors, Henoch and others, have stated that aphasia is common in children after typhoid fever. He wished to know whether the literature showed many instances of real aphasia, or whether the speechlessness was merely a part of the mental disturbance.

Dr. Edsall replied that so far as he could determine from a rather hasty review of the literature relative to the question of aphasia in childhood after typhoid fever that true aphasia itself occurs occasionally, but that in a number of the cases that have been called aphasia the speech disturbance has been only a part of the general stupidity of a dementia or of the depression of a melancholia. In adults one often sees speechlessness that may last for a long time, and may be due to mere unwillingness to speak or to general weakness of intellect, and not to any defect in the speech mechanism itself. One may see the same thing in children. That this was the case in some instances in the literature that were called aphasia, is apparently shown by the fact that in some cases the children, when their moods changed would talk most excessively. Aphasia undoubtedly occurs, but probably somewhat less frequently than has been generally believed.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, December 13, 1904.

DR. J. H. JOPSON, PRESIDENT.

DR. MAURICE OSTHEIMER read a paper on

INCONTINENCE OF FECES IN CHILDREN.

He stated that incontinence of feces alone is very uncommon; incontinence of both feces and urine is rare, though enuresis occurs commonly among children. These cases were divided into those in which some cause for the condition was found, either general or local; and those in which the disorder was due to the general nervous condition of the child. In some of the latter the incontinence persists from infancy; in others it develops suddenly, in apparently healthy children over three years of age. Cases illustrative of these subdivisions were described—twelve from literature and four observed by Dr. Ostheimer. Recovery followed good food, fresh air, tonics and large doses of strychnin. Recurrence is common, whenever the child's general condition is not good.

DR. W. REYNOLDS WILSON read a paper on

HEMORRHAGE OF SYPHILITIC ORIGIN IN THE NEWBORN.

This article will be found in full on page 7 of this volume among the "Papers."

DR. J. H. JOPSON referred to the marked confusion that exists in the literature concerning the various forms of hemorrhage in the newborn, particularly in regard to the point whether the hemorrhage is infectious or is due to syphilis, hemophilia or other causes. Dr. Wilson's paper brought out this point very clearly, and in his classification he had succeeded admirably in differentiating between them.

DR. D. J. MILTON MILLER read a paper entitled

A NOTE ON THE DURATION OF THE PRODROMAL PERIOD IN RÖTHELN.

This article will be found in full on page 16 of this volume among the "Papers."

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Dr. Westcott said he was pleased that Dr. Miller had emphasized the not infrequent existence of a distinct febrile prodromal period in rötheln, as he had encountered it a number of times, and considered it an important aid in distinguishing the disease from scarlet fever of mild type. Personally he had encountered very few cases that had assumed the socalled scarlatiniform type with sufficient mimicry to raise grave question in the diagnosis. When an eruptive disease suggested scarlatina so closely as to give rise to reasonable doubt, he considered it his duty to make the more serious diagnosis, reserving the right to revise it after a due period of observation. Cases of scarlet fever of such mild degree are so frequently encountered that this possibility must be borne constantly in mind, and therefore the wider recognition of the possible occurrence of a prodromal period in rötheln, lasting from two days, as he had repeatedly seen it, to five days, as noted in one of Dr. Miller's cases, offers a valuable point in diagnosis when the rash alone might be viewed with suspicion.

Dr. Miller said that rötheln more frequently than is supposed shows a definite prodromal stage. He also had never seen the scarlatiniform type of eruption referred to by Dr. Westcott, and considered that the rash most commonly resembled measles. The last case mentioned in his paper could easily have been mistaken for measles from the eruption, but the rapid subsidence of the symptoms and the known source of infection made the diagnosis clear.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, January 10, 1905.

DR. JOHN H. JOPSON, PRESIDENT.

CASE OF EXTREME INTESTINAL ATONY.

DR. D. J. MILTON MILLER exhibited the patient, a child of twenty-eight months. The patient had been on the programme for December, as a case of possible abdominal tuberculosis, but further observation had caused a revision of the diagnosis. The child, now twenty-nine months old, was well until its tenth month, when it was weaned and fed upon condensed milk and other proprietary foods. At fourteen months it was separated from its mother and placed in a baby farm, where it remained several

EXTREME INTESTINAL ATONY; PATIENT TWO YEARS AND THREE MONTHS OLD.

months. The mother then claimed it again, and noticed for the first time the abdominal distension and that the mental condition was impaired, whereas formerly it had been as bright as most children of its age; this she attributed to dosing with paregoric, while the little patient was in the farm. Sometime after this it was sent to Atlantic City, and thence to St. Christopher's Hospital, from which place it entered Dr. Miller's service at the Children's Hospital. During these many months it had had almost constant diarrhea, and had been fed most improperly. The child showed distinct signs of rickets, particularly beading of the ribs, square head and patent fontanel; the extremities were quite emaciated, but its most conspicuous feature was the enormously distended abdomen. This measured 22 inches at the navel, and 23 inches midway between the latter and the ensiform cartilage. That the distension was largely intestinal was shown by the failure to out-

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line the stomach when it was filled with water; and that the colon, at least, was dilated by its ability to retain for 20 minutes a quart of fluid. Over the distended abdomen the superficial veins were well-marked, but it could never be determined that they joined with those of the thorax. Neither liver nor spleen could be palpated, nor could enlarged glands nor masses of any kind be discovered in the abdomen. The distension was almost entirely due to flatus, of which the child constantly passed large quantities; fluid had never been detected. The temperature had been constantly above normal (99°-100°F., or 101°F.), and there was a secondary anemia (red blood cells, 5,532,000; white cells, 11,000; hemoglobin, 65 per cent.). The urine contained a trace of albumin, and from time to time a few hyaline and granular casts. Because of the possibility of abdominal tuberculosis (mesenteric lymph nodes or peritoneum) $^1/_{10}$ mgm. of tuberculin was given by injection on two occasions. Prompt reactions followed; at one time the temperature rose to 103.5°F., at the second to 102°F. This gave rise to the diagnosis of possible intestinal or mesenteric tuberculosis; but, as already stated, this opinion had been altered, and was not warranted simply because the reaction was present; it might have been caused by a tuberculous focus elsewhere, the bronchial lymph nodes, for instance, which might well be implicated, as there had been long standing bronchial catarrh. For the same reason tuberculosis of the mesenteric glands could not be absolutely excluded; for, although they did not appear to be enlarged, the chronic intestinal catarrh would render them liable to infection. It seemed very unlikely that the enteritis was tuberculous, the clinical history and signs being quite opposed to such an idea. At one time, the question of congenital dilatation of the colon had been considered; but was dismissed, because the abdomen appeared to have been natural until after weaning at the tenth month, and, although the congenital affection may not reveal itself until later infancy, yet the entire absence of a history of constipation in the patient would seem to exclude that condition. Dr. Miller was inclined, consequently, to regard the case as one of extreme intestinal atony due to rickets and long standing intestinal catarrh, although abdominal tuberculosis could not be positively barred out. The patient's mental condition was interesting; it was far behind healthy children of equal age; it could not talk, and did not seem to recognize its attendants, although long familiar with their presence; it would lie upon its back the live-

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long day playing with its feet or hands, and had a peculiar, almost athetoid, way of moving the fingers about. Whether this condition was due to long-continued mal-nutrition, or to actual arrest of cerebral development, could not be determined. The child had greatly improved while in the hospital, and had gained in two months about 6 pounds. The treatment had consisted of abdominal massage with guaiacol ointment, hypodermic injections of strychninæ (gr.-100 daily), and careful feeding. For a month past it had been under Dr. Griffith's care.

Dr. J. P. Crozer Griffith agreed with Dr. Miller as to the diagnosis. The only other probable condition was idiopathic dilatation of the colon, and in a study of the literature of this disease Dr. Griffith had noted that practically all cases have marked constipation, perhaps interrupted by occasional attacks of diarrhea. The presence of persistent diarrhea in this case makes dilatation seem improbable.

Dr. Miller asked Dr. Edsall what he considered to be the significance of the tuberculin reaction.

Dr. Edsall replied that his personal experience has been rather slight, but from the literature it seems highly probable that the child has some focus of tuberculosis, though there is an appreciable percentage of error in this method of diagnosis.

Dr. Miller had asked the question because of the patient's prompt reaction to two separate injections of tuberculin, which he thought was strong proof of tuberculosis somewhere, possibly of the bronchial or mesenteric glands, as it had suffered for a long period from two conditions liable to give rise to infection of lymph nodes, viz., bronchial and intestinal catarrh.

Dr. J. P. Crozer Griffith reported a case of

CONGENITAL STENOSIS OF THE COMMON BILE DUCT.

This article will be found in full on page 25 of this volume, among the "Papers."

Dr. La Roque said that he had made the autopsy and upon opening the abdominal cavity found the gall bladder enlarged and the intestine in this region deeply stained with bile, while the intestinal contents and mucous membrane were not so colored. After a search over the surface of the duodenum the papilla was found, but the biliary canal was not patulous from the intestinal

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end. Bile could not be forced through the lower end of the common duct and a fine straw could not be passed through from above nor below. For a short distance near the papilla, the common duct seemed to be merely a fibrous cord. The cystic and hepatic ducts were patulous.

Dr. Hamill asked whether the autopsy reports show varying degrees of obstruction. It is difficult to see how cases of complete obstruction could live for any length of time. In a series of cases of sepsis in the newborn that he had studied, there was usually diarrhea with greenish stools, fever, extensive hemorrhages from the mucous membranes and often jaundice of varying degree. In the cases with jaundice he had found no obstruction of the biliary duct at autopsy.

Dr. Hand, in reference to the question whether the stenosis or the biliary cirrhosis is the primary condition, described a case that he saw at autopsy several years ago in a child that died at seven months after having had an increasing degree of jaundice and ascites. He found a very marked biliary cirrhosis with stenosis of the ducts. The latter was evidently the primary condition and the cirrhosis was due to it.

Dr. D. J. Milton Miller asked whether the literature shows that fever occurs in congenital stenosis of the ducts. If so, it must be extremely difficult to distinguish this condition from septic cases. He mentioned as an instance of the latter a child removed by Caesarean section that had rapidly developed purpura, hemorrhages, jaundice, high fever and soon died.

Dr. J. P. Crozer Griffith said that fever is not a symptom in obstruction. If the child lives long enough ascites may develop as a result of the liver becoming atrophic. This, however, is unusual. Seven months is a long time for the condition to last, but it has been known to last as long as eight months. Complete obstruction must, of course, be fatal; and in such a case the obstruction may be very localized, or may extend over a large part of the ducts, and the gall bladder has even been entirely absent. In some cases, however, it is highly probable that the obstruction is not complete at first, and its effects are but slight at first, and that they ultimately grow worse and finally cause death. There are cases unusual in which obstruction has taken place after several years, due possibly to a recurrence of a cholangitis which developed originally in fetal life, but which for some reason has not advanced to a

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condition of complete obliteration. We have no knowledge how often this occurs.

DR. HOWARD CHILDS CARPENTER reported

A CASE OF LATERAL CURVATURE IN AN INFANT ELEVEN MONTHS OLD.

The family history was entirely negative, with the exception that the mother has had four miscarriages, and three premature births. This child was born at term in an easy labor, lasting one hour. It had no deformity at birth. It was fed exclusively on the breast until six months old, since then, besides the breast, milk and crackers, meat broth and mashed potatoes. It has *never* been sick with one exception: an attack of enteritis when six months old. The mother denies all history of traumatism. She carries the child as much on one arm as on the other.

Ten days ago was the first time that the mother noticed anything wrong with the child's back. The child has no subjective symptoms whatever. No pain or tenderness. It has three teeth, and its present weight is 13¼ pounds. It is poorly nourished, and its muscular development is below par. The child has a well-marked primary right dorsal scoliosis, with rotation of the bodies of the dorsal vertebræ. There are secondary compensatory lumbar and cervical curves. The lateral curvature is of the third degree. There are no evidences of rachitis.

DR. D. L. EDSALL and DR. C. W. MILLER read a paper on

THE DIETETIC USE OF PREDIGESTED LEGUME FLOUR, PARTICULARLY IN ATROPHIC INFANTS,

with observations on absorption and metabolism. Bean flour and other legume flours have for many years past occasionally been used by members of the profession as well as by the laity in small amounts in feeding infants. Relatively little attention has been paid to this, however, and there has been no investigation of the actual suitability of such foods for infants. Unless predigested they must be difficult of digestion and it is impossible to give more than a very small quantity. The present study was undertaken for the purpose of determining by accurate observations to what extent predigested bean flour can be successfully given to infants, and how far it may be possible to use the nitrogenous constituents of bean flour in place of milk proteids, for it would seem to be readily possible to introduce as much as 1 per cent. of proteid from this source into a baby's food, and, if necessary, even much

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more. The results obtained led to an investigation of the influence of bean flour on metabolism and to the conclusion that it is not improbable that it has some special influence favoring tissue building processes in atrophic infants. The results of three absorption experiments and one metabolism experiment in infants, and of a metabolism experiment in an adult showed that the bean proteid is absorbed very well indeed and that its influence on metabolism is at least equal to that of milk, and perhaps more than equal in this special class of cases. The starchy portion of the flour only was predigested and after this was done the bean flour solution was added to a milk mixture.

The clinical results in 28 cases, only 15 of them having been satisfactorily studied, however, were detailed. The results as to general improvement and gain in weight were very striking, the babies in nearly all instances gaining very rapidly, while previously they had been stationary or losing. The series, however, is still a small one and the results may be partly due to chance. So far as the study has been carried the results seem extremely encouraging as compared with those obtained by other methods of feeding atrophic babies in hospitals.

Dr. J. P. Crozer Griffith said that he had for a number of years been using freshly malted cereal gruels in certain cases, in the effort to avoid entirely the employment of any of the commercial malted foods on the market. Accident drew his attention to the question of the values of bean flour as a food, and he had urged the investigation of this upon Dr. Edsall. That explained his connection with the matter. The credit of the investigation rested entirely with Dr. Edsall. The great value of Dr. Edsall's results was not only that they prove clinically that children would often thrive remarkably upon a food mixture containing malted bean flour, but that they established scientifically the value of the vegetable proteid contained in it as a substitute for that of cow's milk, when the latter was found to be difficult of digestion. The investigation, by modern scientific methods, showed that the proteid was easily digested and assimilated. This was something which, as far as he knew, had never been done before. It was a very important addition to our knowledge of the dietetics of infancy.

Dr. D. J. Milton Miller said that any method of feeding atrophic infants that proves to be at all successful would be ex-

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tremely valuable, and, therefore, he welcomed this addition to our armamentarium. He asked whether the infants would not be likely to develop scurvy on this food. He also desired to know whether any of the cases treated were acute cases, and, if so, whether the stools had been blackened by bismuth and whether there was much flatus, from the formation of sulphuretted hydrogen, which the legumes were well known to produce. For many years he had observed that as soon as bismuth began to blacken the stools in the acute enteritis of infancy, it was a sign that the case was beginning to improve; this he had attributed to the appearance of sulphuretted hydrogen, and a return to more normal conditions. It would be interesting to know whether bismuth administered simultaneously with legumes in similar cases would be converted into the black sulphid from the very first.

Dr. Hamill said that one great advantage of this method of feeding seemed to him to be that it enables one to use enough milk to protect the child from the development of scurvy.

Dr. C. F. Judson said that he had come across the following remarkable formula for vegetable soup in a French medical journal, recommended by Méry in chronic digestive disturbances in childhood where milk has to be temporarily excluded from the diet. Boil 400 gm. of carrots, 300 gm. of potatoes, 100 gm. of turnips, 80 gm. of dried peas and beans, and 35 gm. of sea salt for four hours in seven quarts of water, and carefully strain the bouillon. The soup must be prepared fresh every day.

Dr. Edsall, in closing the discussion, said that none of the cases had acute symptoms when given bean flour, though several were convalescent from acute attacks. The points about the preparation that make it worthy of investigation are, that it is not proprietary, that it furnishes a means of increasing the proteid when it is difficult to do this with milk, that it seems to be easily digested and usually causes the digestion to improve in subacute cases, and that it can be given with as much milk as seems suitable in the individual case, thus avoiding, in large part at least, the disorders of nutrition, such as scurvy, that are likely to follow the exclusion of milk from the diet. It is also of importance that the food could be used exclusively, as a more or less temporary expedient, the amount of nitrogen in the bean flour alone being entirely sufficient for all purposes. This had been done with great success in one desperate case. It is the nitrogen content of the legumes that gives them their great value.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, February 14, 1905.

DR. J. H. MCKEE, PRESIDENT.

A CASE OF HEAD NODDING ASSOCIATED WITH NYSTAGMUS.

DR. R. S. MCCOMBS presented this patient, an infant four months old, with associated movements of the head and eyes. The family history was negative. The child's birth was normal, and he had been well ever since. He had been breast-fed exclusively. The nystagmus was the first symptom noticed by the mother, when the patient was about three and one-half months old. Shortly after this the head nodding appeared. The child appeared fairly well-nourished. There was noted a rachitic rosary, and epiphyseal enlargement at the wrists. There was a constant nodding of the head, anteroposteriorly, with an occasional retraction to the right side. There was also present a vertical nystagmus, equal on both sides, and increased by near fixation. The fundus was normal. The interesting features in connection with this case were: The early age of the patient, the absence of an exciting cause, with the exception, perhaps, of the rickets, and the fact that the nystagmus was vertical.

DR. SPILLER said that the cause of these involuntary movements is unknown. Such movements are frequently seen in children with rachitis and not infrequently disappear later. It is difficult to find the explanation in the rachitis. As regards the view that these movements are caused by cortical irritation, it is to be acknowledged that the centre for the movements of the head, and probably that for the associated movements of the eyes, are near together. Involuntary movements of the upper limb are not commonly associated with these tremors of the head and eyes. If the cause were cortical irritation, it is remarkable that the upper limb usually is not affected. The centre for the upper limb is near the centre for the face and near the centre for the associated ocular movements in the frontal lobe.

A CASE OF PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

DR. H. C. CARPENTER presented this patient, a boy of fifteen years. The weakness of the lower extremities was first noticed when he was about four years old, and since then it has been increasing steadily. The patient presents all the classical symptoms of the disease, with typical station, gait and pseudomuscular hy-

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pertrophy. There is marked hypertrophy of the calves, also of the tibialis anticus, quadratus lumborum and infraspinatus muscles. The scapulo-humeral muscles are wasted. There is also mental weakness. The knee jerks are not diminished. No reactions of degeneration present in arms or legs. A full report of the case, together with photographs, appears in this volume, among the "Papers," page 32.

DR. SPILLER said that he recently had seen a case of muscular dystrophy which showed atrophy of the bones, and that the latter is often overlooked in the study of this disease. He had reported the microscopic examination of a case originally studied by Duchenne, of Boulogne, in which the nervous system was found to be normal. There was intense atrophy of all the muscles, even of the diaphragm. A few cases have shown lesions of the nervous system, but muscular dystrophy is essentially a disease of the muscles.

DR. BOCHROCH said that he was unable to elicit any knee jerks in this patient, although he understood Dr. Carpenter to say that this sign was present. The knee jerks are generally absent; at least it is unusual to find them in a case so far advanced. He recalled a number of instances of the disease occurring in several members of a family. This patient, he believed, was the only one of his family so afflicted.

DR. CARPENTER said that the knee jerks were present in this patient, if not exaggerated. The notes of the case from the Children's Hospital showed that the knee jerks were exaggerated when the boy was a patient in that institution.

A CASE OF CEREBROSPINAL FEVER.

DR. GRIFFITH, in presenting this patient, said that there is much discussion regarding even the existence of cerebrospinal fever as an entity. While some text-books give it a place among the infectious diseases, others do not refer to it at all in that connection, and one must seek for it among the various forms of meningitis. There seems to be no question that the same complex symptoms may be produced by different varieties of germs, the meningeal disease being often secondary to some other affection. It is also true, however, that leptomeningitis does present itself in an epidemic form, and with both the fixedness and the power of variation of symptoms which are so characteristic of infectious diseases.

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By those who regard cerebrospinal fever as an independent infectious disease various types are described. Among these may be mentioned the typical, the abortive, the intermittent, the chronic, etc. One of the most trying of these to the patience of the physician and of the family is the chronic form, and as a good instance of this, showing how prolonged a course the affection often runs, he wished to exhibit the patient whom he had brought before the Society to-night.

Willie Rodosky, six and one-half years old, was brought to the Children's Hospital, Philadelphia, on November 11, 1904, where he was under the care of Dr. Miller. There was nothing of moment in the family history, and the patient had been a healthy child. Five days before admission he was taken ill with fever, headache, vomiting, constipation, abdominal pain, anorexia and malaise. On the evening of the second day he became delirious, and had continued so at times. A day or two before admission convulsive twitchings had occurred, and his neck became stiff. On the fourth day the eyes became crossed. Abdominal pain was at times complained of. On admission into the hospital he was irritable and semidelirious, but not unconscious; the eyes were crossed, the pupils were slightly dilated, but reacted to light. There was cervical opisthotonus, great rigidity of the whole body, and extreme hyperesthesia, so that touching of any portion of him caused him to utter a sharp cry. Kernig's sign was present. The leukocytes numbered 19,520.

The patient continued in much the same condition, being very irritable, fretful and hyperesthetic. Examination of the eye-grounds, on November 12th, gave practically negative results. On this date lumbar puncture was done, and 2 drams of cloudy fluid removed. On sedimentation a creamy yellow deposit of pus formed. Examination showed the presence of the diplococcus intracellularis.

Improvement was extremely slow. Gradually retraction of the head diminished. A second lumbar puncture done on the 16th gave a similar fluid, so thick, however, that it was necessary to aspirate it. By the end of the month some improvement had shown itself, the boy being quite rational to questions and less fretful, though rigidity and hyperesthesia continued, and in a few days retraction had shown itself. Throughout December there was very little alteration in any symptoms. Examination of the eyes on January 24th, by Dr. Randall, showed small extravasations of blood in

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each retina, and the general appearance of an albuminuric retinitis. During the last week of December he was out of bed in a chair, although very weak and thin. Throughout January he improved steadily, although slowly, having gained the power to walk with an unsteady gait. Both eyes could be turned to the right. On the 30th of the month he had nausea and vomiting, followed by what appeared to be an attack of fainting, but which was probably a slight convulsive seizure. Examination of the blood on the following day showed 14,350 leukocytes. In the week following this attack he deteriorated in health, and lost over 2 pounds in weight. On the 13th of the month two other convulsions occurred, this time more marked. The blood now showed 17,050 leukocytes, the differential count giving 13 per cent. of leukocytes and 81 per cent. of polymorphonuclear cells.

The boy has a normal expression, except for the internal strabismus. He is thin, and looks rather feeble and delicate. The interesting feature is the temperature chart. Some days after admission the temperature ran a course of 101° F. to 104° F., without any definite regularity. It then gradually descended, and toward the end of the month would sometimes touch the normal line, although generally about 102° F. Throughout December it was extremely irregular, some days not exceeding 100° F., but generally going to 102° F., and occasionally 103° or 104° F. About the middle of December it exhibited an intermittent type, without any decided regularity. It is to be noticed, however, that until nearly the end of January the temperature did not become afebrile. Even then it showed anything but a regular line, being often below normal, and other times reaching 100° F., with occasional rises to 101° F. During the last week of January there was a still more irregular temperature curve, of which 100° F. might be taken as the middle line, the temperature going irregularly above and below this.

It is now at the 100th day of the disease, as near as we can calculate. Were it not for this temperature chart one might think the boy had practically recovered. We have, however, three other factors which make me uneasy about him. The first symptom is the strabismus, although this I imagine to be but the remaining effect of some lesion, the active state of which has probably passed away. The second is painful rigidity of the neck, and at times of other parts of the body. The third symptom, and a grave one, is the development of convulsions. These three symptoms, com-

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bined with the irregular fever, give us every reason, I fear, to believe that we are not yet through with the inflammatory process, or with the damage which the attack may leave.

Note, March 7th.—It is now three weeks after the exhibition of the patient, and the 121st day of the disease. The rigidity of the neck and the irregular temperature are still present. The strabismus is decidedly less. There have been no more convulsions.

Dr. Miller said that he had seen the patient before at the Children's Hospital, where he had been under his care for a month. He thought he had improved very much, for when he last saw him he was still in bed and emaciated. He considered the case interesting from the fact that, notwithstanding the notes, the child had never been very delirious, and after the first ten days became quite rational, answering questions and laughing when spoken to. He was extremely hypersensitive to touch or sound; his neck was very rigid, and he could not move at all. Dr. Miller thought the best treatment in these cases was opium; it had a very excellent effect on this patient. The thermo-cautery, also, had been used in this case without benefit. Lumbar puncture was resorted to three times; 2 drams being withdrawn at the first tapping, and half an ounce upon each other occasion. This measure gave no relief, nor had he ever known it to be of appreciable benefit in the cases in which it had been employed by him. A point of differentiation between the epidemic cases, and those due to other organisms, was that many of the former get well or become chronic, while the latter are nearly all fatal, at least, such cases at the Children's Hospital had all died. Dr. Miller thought there was not sufficient evidence for assuming that this form of meningitis is contagious, and that the rule of the Board of Health requiring them to be reported was unnecessary. Often they cannot be distinguished, clinically, from the other forms of meningitis.

Dr. McKee called attention to the occurrence of hydrocephalus occasionally following cases of chronic epidemic meningitis. In a series of cases with this complication, recently reported, one had vomiting, fainting spells and later convulsions. He thought that from the symptoms such a condition might subsequently appear in this child.

Dr. Griffith called attention to the fact that deafness, blindness, and even idiocy, often follow this disease. Thus, for in-

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stance, of two sisters ill in the Children's Hospital, one died and the other left, apparently entirely well; but to their disappointment was brought back some months later totally deaf. He thought the child which he presented this evening was by no means yet out of danger, as there was evidently some active process still existent.

A CASE OF MULTIPLE EXOSTOSES.

DR. GEORGE W. NORRIS presented this patient, a boy of seven years. The family history was negative. He has had pertussis and measles, and two years ago had scarlet fever, as a sequel to which he suffered from what appears to have been an attack of arthritis, which confined him to bed for six weeks. About eighteen months ago the father noticed that the boy had "queer joints." There has never been any complaint of pain since the attack of arthritis. The child was brought to the dispensary on account of failing health, pallor and frequent attacks of bronchitis. His temperature was 99⅕°F.; pulse 90. There was noted broncho-vesicular expiration with slight impairment of resonance, posteriorly, at the right apex, but no râles. Distributed over the skeleton were numerous osteophitic excrescences, painless, and causing no restriction of motion. They were symmetrical and bilateral, being most commonly felt on the long bones, near the epiphyseal junctions; some on the epiphysis, others on the shaft. They were more or less conical in shape, the base being nearest the skeleton. They were noted in the following regions: The upper ends of both humeri, the one on the right about the size of a small lemon; on the external surfaces of both radii and ulnæ, the size of chestnuts; the clavicles, the tibiæ and fibulæ, the upper end of each femur and the costochondral junction on the left side from the fourth to the seventh ribs.

DR. ELEANOR C. JONES stated that she had at present under her care a family of four children all of whom had multiple exostoses. The father is also afflicted with the disease. The youngest child is a baby of eighteen months.

DR. EDSALL recalled a similar case in a boy who died of pneumonia when fourteen years old. The disease first appeared in the scapula, when the child was two years old, and the condition was said to have been diagnosed and treated at that time as a fracture. This patient had only half a dozen exostoses, but they were all very large; one on the thigh was as large as an infant's head.

DR. W. J. TAYLOR knew a man forty-five years old who had

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eight or ten exostoses. The disease was an inheritance from his father. One exostosis on the thigh was so very sharp that it would frequently pierce the muscle and cause an acute inflammation. Recent studies have shown the disease to be closely related to myositis ossificans.

A CASE OF HYDROCEPHALUS.

DR. J. H. MCKEE again presented a patient with hydrocephalus, an infant shown before the Society just one year ago. In November, 1903, when pressure symptoms were marked, 23 ounces of blood-stained fluid were removed by puncture of the left lateral ventricle. The fluid reaccumulated slowly, and the child was tapped again in March, 1904; 11 ounces of blood-stained material being secured. Blood was found by microscopic study, and 6 or 7 tubercle bacilli were revealed in each microscopic field. Since that time the boy has improved in every way, and the fluid has not reaccumulated. Indeed, the cranial circumference has decreased 1 inch, from 26¼ to 25¼ inches. The sutures have also closed, and the fontanel has become depressed and soft. Intelligence and physical development have also progressed most remarkably. Recently the boy has suffered from epidemic influenza, double otitis media and an attack of facial erysipelas, in spite of which his meningeal symptoms have not recurred.

DR. SPILLER said that internal hydrocephalus occasionally results from meningitis, and is caused by the occlusion of the communications between the subarachnoid space and the fourth ventricle. It is difficult to determine the origin of hydrocephalus in certain cases. He had been able in 1 case to find the cause in occlusion of the aqueduct of Sylvius by proliferation of neuroglia, and in another case by an inflammatory process about the foramen of Monroe of one side. In the first case the lateral and third ventricles were much distended and the fourth ventricle was normal. In the second case the internal hydrocephalus was confined to the lateral ventricle of the side on which the foramen of Monroe was partially occluded.

DR. MCKEE said that he had examined a woman who was an intimate friend of the mother of this child, and who resided in the same house with them, and found her to have tuberculous disease of the right apex. She was accustomed to take care of the baby in the early days of his life. He thought this might throw some light upon the etiology of this case.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, March 14, 1905.

JAMES H. MCKEE, M.D., PRESIDENT.

A FAMILY OF FIVE CHILDREN WITH MULTIPLE EXOSTOSES.

DR. ELEANOR C. JONES presented these patients. Five of the eight living children are so afflicted, and the father, who died at the age of forty years, also had numerous exostoses. Joseph, thirteen years old, and Eddie, eight years old, have each about fifty of these growths. The three other children, including a baby of eighteen months, have only from one to three exostoses each. Joseph has also a congenital shortening of the bones of the right forearm.

DR. MILLER said that there is possibly a similarity in these cases of multiple exostoses to those of chondrodystrophy. (He was about to show a patient with this latter disease.) But the similarity may be only apparent. In chondrodystrophy, however, the lesions are symmetrical and bilateral. There is a failure of growth of the cartilage cells of the diaphyses, the periosteum alone producing new bone, and a shortening of the shaft results in consequence. In two of Dr. Jones's patients there is an unbilateral shortening of the bones of an upper extremity, with great overgrowth of the bone, and he thought that the two diseases might be allied, especially as the condition in both diseases is congenital.

CHONDRODYSTROPHY FETALIS.

DR. D. J. MILTON MILLER presented this patient, a boy seven and one-third years old. The parents were both healthy and well-formed; no history of maternal impressions, nor of anything bearing upon the causation of the condition could be elicited. Birth had been instrumental, and the large head and small limbs were noted at this time. The boy presented the characteristic features of the disease: normal trunk; large head, with depressed bridge of nose; deformed and shortened extremities; short, fat fingers, of equal length, with characteristic separation of the middle and ring fingers; and marked lordosis. Measurements, compared with those of a normal child of equal age, illustrated in a

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striking manner, that the dwarfed stature was entirely due to the diminished size of the lower extremities. Radiographs showing the changes in the cartilage and bones characteristic of the affection were also exhibited.

DR. MILLER said that the etiology of chondrodystrophy is very obscure. There was nothing in the family history of this patient to account for the disease. Two other children are in good health, and the father and mother are well and strong. In chondrodystrophy there is a failure, or irregular growth, of the cartilage cells of the diaphyses; in addition there is a fibrous ingrowth of the periosteum between the epiphyses and the diaphyses. Growth of the bone is consequently impossible.

STUPOROUS INSANITY AT PUBERTY.

DR. CHARLES W. BURR showed this patient, a boy fourteen years old, who was admitted to the Philadelphia General Hospital in September, 1904. His mother gave the boy a bad name, saying he was profane, a cigarette smoker and stayed out late at night with his father, who is a huckster. In July, 1904, after an attack of constipation lasting two or three days, he began to act strangely. He would take off his clothes at any hour of the day and wander aimlessly about the house. Soon he became sleepless, and would walk up and down stairs all night, giving no reason therefor. Still later, he accused his mother of poisoning him, refused to eat and finally talked only in monosyllables and only when spoken to.

On admission he was well nourished, seemed stubborn and talked little. He said he had pains all over the body. He was very emotional, and cried a great deal. He spat a great deal, and his brows were always contracted. The pupils were widely dilated. The knee jerks were absent. The eye grounds were normal. In the early part of November he became motionless and irresponsive to all stimuli. When pricked deep in the arm or leg he would wince, but showed no other sign of pain. He paid no attention to anything about him, and passed urine and feces in bed. He was mute. For a little while he was slightly cataleptic, and at another time resisted passive movements. He lost flesh rapidly. The temperature had been normal throughout. He would make no effort to chew or swallow food put in his mouth, and was fed by a medicine dropper. On admission the blood was normal, but later the hemoglobin fell to 52 per cent., the number

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of corpuscles remaining normal. He began to improve on January 3, 1905, when for the first time he swallowed a little milk willingly. Since then he has slowly become somewhat better. He now talks a little, will willingly chew and swallow soft food, indicates when he wishes to micturate, and obeys simple commands. He has put on a little flesh. Taking him as he now is, it would be difficult to determine whether he is insane or hysterical. With the history, the diagnosis is clear. The mode of onset is characteristic of one of the types of pubertal insanity, or, as it is now fashionable to call it, dementia precox. Dr. Burr was inclined to believe that the outlook is rather good.

Dr. Rhein asked Dr. Burr why he had not called the condition dementia precox; and also upon what he had based such a good prognosis. He thought that the symptoms exhibited by Dr. Burr's patient, who at one time complained of delusions of persecution, again of catalepsy, and still later of stupor, warranted a diagnosis of dementia precox.

Dr. Burr said that there was no objection in calling the case dementia precox, but in his opinion it was preferable to call it pubertal insanity. The term dementia precox has been made to include so many things that it is fast becoming a very indefinite term. He had given a good prognosis for the reason that the boy has already improved both mentally and physically, and is improving daily.

A CASE OF EXTENSIVE HEART DISEASE, APPARENTLY WITH PERFECT COMPENSATION.

Dr. Frederick Fraley presented the patient, a girl of ten years.

A CASE OF FATAL CHOREA.

Dr. J. P. Crozer Griffith reported a case of fatal chorea, in which the death appears to be the direct result of the violent muscular action. The child was unable to speak, and later became delirious and finally comatose. There was a mitral systolic murmur present. Autopsy showed the cardiac lesions of endocarditis, with microorganisms in the muscle and the mitral leaflet.

Dr. Burr said that about fifteen years ago he made an autopsy with Dr. Griffith upon a child that had died of chorea. Endocarditis was present, as it has been in almost every, if not every, case of chorea that has come to autopsy. He thought it was im-

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possible to determine whether Dr. Griffith's patient died from the infection or from the violence of the movements.

Dr. Rhein said it was unfortunate that the brain and the cord had not been obtained in Dr. Griffith's case. The nervous system has been studied microscopically in a few cases of chorea, but nothing very definite has been described. Some observers have looked upon the chorea bodies as being characteristic. Within the last year Dr. Rhein had examined the brains of two children who had died during chorea. A capillary hemorrhage was found in one case, but otherwise the findings were negative. In the second case there was some round cell infiltration in the cortex, and some perivascular dilatation, besides a moderate amount of infiltration of the pia. The blood vessels were numerous and distended, especially in the white matter posterior to the corpus callosum. These cases will be reported in detail later.

Dr. Eshner recalled 2 cases of fatal chorea that he had seen. One was a boy of seventeen years, who came under observation with rheumatic symptoms and who afterwards developed chorea characterized by violent movements. No lesions were found in the brain and cord at autopsy on both macroscopic and microscopic examination. In the second case the cause of death was exhaustion following pericarditis and endocarditis with nephritis. In another case, not fatal, there occurred a series of convulsions, resembling those of uremia or eclampsia, together with high fever. The violence of the movements in cases of chorea like those under consideration would seem to lend support to the view according to which the disease is considered infectious, the secondary intoxication in these instances being especially intense.

Dr. Miller had seen Dr. Griffith's patient at the Children's Hospital. The boy had a very septic appearance, and he thought that some infection apart from the chorea might have been a contributory factor in his death. Ordinarily, cases of severe chorea, even with complications, recover. He mentioned the case of a girl of twelve years who passed through a severe attack of chorea, complicated by pneumonia. She recovered from the chorea, but developed empyema and parotitis and subsequently died.

Dr. Griffith thought that the case Dr. Burr referred to was

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that of a woman who died at St. Agnes' Hospital some years ago. That patient was the only other fatal case of chorea he remembered ever having seen. Her movements were not specially violent, and she died from endocarditis. He thought that the jactitation of such cases as the one now reported could well be described as terrifying. Although this boy's appearance was septic, no focus could be discovered to account for this condition, and although it is perfectly possible that the death was due to the endocarditis or the septic condition, yet the impression given was certainly that of death from exhaustion, the direct result of the violent muscular action.

NECROTIC STOMATITIS FOLLOWING MEASLES AND PNEUMONIA.

Dr. R. Max Goepp reported the case of a child eighteen months old who developed, during the course of a bronchopneumonia complicating measles, a necrotic patch at the left angle of the mouth and confined to the inner surface of the cheek. The necrotic area was one and one-half inches long and one-quarter of an inch wide. At this time the child was convalescing. Constitutional symptoms were not marked; the throat was clean. Free cauterization under chloroform anesthesia was performed first with nitric acid, and later the paquelin cautery was applied, supplemented by curettage. Diphtheria organisms were found in both the throat and mouth; the cultures showing in addition staphylococcus albus and streptococcus pyogenes. No bacilli nor higher fungi were observed. No antitoxin was administered. Recovery followed without deformity.

Dr. Levi said that he had seen two patients with Dr. Sailer who had noma following typhoid fever. The Klebs-Löffler bacillus was obtained in pure culture, and although antitoxin was given there was no appreciable benefit from it. One of the children died.

Dr. Jopson said that although he had never obtained the Klebs-Löffler bacillus in the cultures from any of his cases, still he believed there was sufficient evidence at hand to justify the opinion that it is sometimes the cause of this condition. He also thought that the actual cautery or excision is the best treatment in these cases, and antitoxin in doubtful cases.

Dr. Griffith agreed that cauterization is the best procedure in these cases. He was not sure that some of these cases diagnosed

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noma should be classified as such. They represent a condition midway between ulcerative stomatitis and true noma. He considered the term necrotic stomatitis used by Dr. Goepp a very good one for this condition.

DR. HAND referred to an epidemic of necrotic stomatitis that occurred at the Children's Hospital during his residency. The epidemic followed measles and whooping-cough and was much worse than ulcerative stomatitis, but not with the characteristics of true noma, although cauterization was necessary in some of the patients; one case resulted fatally, the infant having had a hemiplegia following whooping-cough.

A CASE OF MALIGNANT SCARLET FEVER.

DR. GOEPP also reported this case. The patient was a girl four and one-half years old who developed high temperature, a rapid and feeble pulse and convulsions, the condition terminating fatally in forty-eight hours. No rash was present. The diagnosis of malignant scarlet fever was made after the appearance of 2 cases of typical scarlet fever in the same household.

DR. HAND spoke of the rarity of malignant scarlet fever and of its two types, one with a decided rash easily diagnosed, and the other with no rash, the diagnosis resting upon the history of exposure, or, as in the present case, on the occurrence of the disease in other members of the family. In his own experience this is the only instance of malignant scarlet fever, all the other fatal cases, two in number, which he has seen, succumbing to a prolonged septicemia.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, April 11, 1905.

JAMES H. MCKEE, M.D., PRESIDENT.

CEREBELLAR TUMOR.

DR. CHARLES F. JUDSON presented this patient, showing the characteristic symptoms of cerebellar tumor. G. T., of Irish parentage, seven years old, male, two months after a mild attack of scarlatina in August, 1903, developed the typical picture of brain tumor involving the posterior fossa, nausea and vomiting, headache, vertigo, staggering gait, failing eyesight, etc. On admission to the hospital in September, 1904, he could stand only with feet widely separated. In walking toward an object he deviated to the right or left, rotation of the body caused marked vertigo, but there was no ataxia in the recumbent position. His knee-jerks were increased on both sides, especially the right, the Babinski reflex was present; there was no ankle clonus. The mind was clear and the memory good; muscular power was not impaired; the cranial nerves were not involved with the exception of the optic. Vision was markedly reduced, double choked disc with beginning atrophy and small retinal hemorrhages being found. No real nystagmus existed, but the eye muscles were paretic on executing extreme movements. Stereognosis was normal, as was the muscular sense. As presented to the Society, the boy was markedly paretic, both lower extremities were moved with extreme difficulty, the right scarcely at all; the upper extremities were less involved, as were also the muscles of the trunk, and here again the right side of the body and right arm had suffered most. The grip of the right hand was feeble, that of the left hand fair. Spastic rigidity was noticed in both legs and the right arm, and a coarse tremor followed on attempts to grasp objects with the right hand. Extreme deviation of the eyes resulted in coarse, oscillatory nystagmoid movements. The pupils were dilated, but responded to light and accommodation. Vision in the right eye was almost gone. There were incontinence of urine and feces, marked vasomotor disturbances, rapid pulse and

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respiration and extreme hebetude. The interesting points in this case were the long duration and gradual course of the disease, and the absence of any definite localizing symptoms such as might have warranted surgical interference, while typical symptoms of brain tumor had been present at a very early stage. From the boy's appearance and family history it seemed likely that the growth is tubercular.

A CASE OF CHYLIFORM EFFUSION INTO THE LEFT PLEURA AND HODGKIN'S DISEASE.

DR. D. J. MILTON MILLER exhibited this patient, a boy of eleven years. The course had been quite rapid; the enlarged glands were first observed fifteen months ago (December, 1903), and had so grown in seven months that they were removed last July, 1904, at the Jefferson College Hospital. Dr. Coplin reported the histology as essentially that of Hodgkin's disease, namely: increased lymphadenoid tissue; proliferation of endothelioid cells; presence of uninuclear and multinuclear giant cells; thickening of reticulum; eosinophiles in moderate numbers; no tubercle bacilli. Shortly after this the glands again enlarged; now on both sides of the neck. The abdomen and feet also began to swell, so that the boy could not get about. This caused his admission to hospital six weeks ago. He presented then practically the same conditions that he does now. There is a mass of enlarged glands on the left side of the neck, and a similar enlargement, although to a less degree, on the right side posteriorly; the axillary and inguinal glands are slightly enlarged. The abdomen contains fluid, and its superficial veins are prominent; the liver and spleen are enlarged. The superficial thoracic veins are distended, especially on the left side, and appear to anastomose with those of the abdomen. On admission there was some fluid in the left pleura. This increased rapidly, and was removed by aspiration on March 4th, and again, because of reaccumulation, on April 6th. The fluid withdrawn was peculiar, resembling skimmed milk (chylous or chyliform). It contained albumin (decided reaction); a trace of sugar; a specific gravity of 1009-12; of alkaline reaction; the cellular elements composed almost entirely of lymphocytes, with a few polynuclears and red blood cells; fat, in small amount, chemically, but not microscopically: cultures and smears sterile. Because of the displaced medi-

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astinum (from the effusion), dullness cannot be demonstrated in the upper sternum or in the interscapular space, nor does the x-ray reveal the presence of a growth in the mediastinum. The urine contains a trace of albumin; a few hyaline casts; no sugar. Several blood counts have been made; the red cells average 4,000,000, the whites 10,000 to 14,000, the hemoglobin 60-70 per cent.; differential count, 90 per cent. polynuclears, 2 per cent. small lymphocytes, 2 per cent. large lymphocytes, 0.5 per cent. eosinophiles. Fever has been almost constant (102-3° F.). The patient is anemic, and the legs and scrotum edematous. He is weak and losing ground and is probably destined not to live much longer.

The diagnosis, Dr. Miller said, was undoubted, viz., Hodgkin's disease, and of a rapid, and, apparently, fatal type. In presenting a case of Hodgkin's disease to the Society three years ago, Dr. Miller maintained that the affection had a definite clinical picture and course, quite different from that of lymphatic tuberculosis, and was a distinct clinical entity; and now Reed, Longcope and Simmons have shown that it has, also, a definite pathological picture, and is a distinct pathological entity. Dr. Miller referred to the large number of lymphocytes in the pleural fluid, as a possible indication of a secondary tuberculous infection; a not uncommon end in Hodgkin's disease. Of very great interest was the chyliform pleural effusion, as the milky appearance was not due to genuine chyle, although its presence could not be absolutely excluded. Effusions of this nature are rare, and are usually divided into chylous and chyliform fluids, or those in which the opalescence is due to admixture of chyle, and those in which it is caused by fatty degeneration of endothelial and epithelial cells; in both cases the fluid is essentially a fatty emulsion. In some cases, however, fat cannot be demonstrated, or is present in too small quantity to form an emulsion, and the milky appearance of the fluid is attributed to globulin, or some form of altered albumin. This, Dr. Miller thought, to be the explanation of the color in his case. The effusion contained but a small amount of fat, only 0.2 per cent, according to Dr. Edsall, who had examined the fluid. Dr. Edsall did not think that agitation with ether had any appreciable effect, but Dr. Coplin found that it cleared the fluid slightly. The latter, also, ascertained that, on treating the sediment (centrifugated) with osmic acid, a small amount of molecular fat could be demonstrated. On the

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other hand, careful microscopic examination failed absolutely to discover fat, and Dr. Edsall could separate but 0.2 per cent., and, in addition, on precipitating out the albumin, found that the opalescence disappeared entirely; hence, he concluded that the milky color was probably due to altered albumin in some form, or in some peculiar combination. The majority of chyliform effusions are associated with neoplasms, and the case presented did not depart from the rule, in that there was probably a growth (lymphadenoma) in the mediastinum or pleura.

CASE OF HODGKIN'S DISEASE WITH CHYLIFORM EFFUSION INTO LEFT PLEURAL CAVITY.

HODGKIN'S DISEASE.

DR. SAMUEL McC. HAMILL presented two patients; the first being a boy (P. J.), aged six years, whose family and previous history were good. The disease was of three years' duration, and first manifested itself by the appearance of a small lump just beneath the mastoid process on the left side. Since its beginning, it has persistently increased in size until it is now about as large as a hen's egg. Gradually most of the glands of the deep cervical chain on the left side have become enlarged, and a few of the post cervical glands on the same side have reached the size of a pigeon's egg. One of the deep cervical glands immediately above the clavicle has attained the volume of a large walnut.

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The development has been gradual and persistent. The glands have never temporarily decreased in volume and there has never been any tendency to suppuration. They have never been painful or tender.

The boy's general health has been excellent. He was presented for treatment with the hope of having the glands removed, in order to improve his appearance. He was not thought to have been feverish. Physical examination showed him to be a well nourished child, with moderate anemia, the blood examination showing 70 per cent. of hemoglobin (Fleischl); 4,090,000 erythrocytes; 8,600 leukocytes. The differential count of the white cells showed:

Polymorphonuclears	53.6	per cent.
Small mononuclears	39.6	" "
Large mononuclears	2.4	" "
Basophiles	0.8	" "
Transitionals	3.6	" "

Careful examination revealed no enlargement of any of the other superficial glands of the body, and no evidence of enlargement of the mediastinal or retroperitoneal glands. The spleen was not palpable; the liver extended two fingers' breadth below the costal margin. On the several occasions on which he had been seen in the dispensary, at the Howard Hospital, his mouth temperature had been between 99° and 100° F.

The second case was (V. S.) a boy of eleven years, whose family history was good, and whose previous history included an attack of measles at the age of two years and scarlet fever at three years. Aside from this, his health was excellent until the beginning of the present trouble.

Three and a half years ago, there was noticed a swelling the size of a marble in the same position as that indicated in the previous case, which gradually increased in size, together with a varying degree of involvement of all of the deep cervical chain and a few of the post cervical glands.

The gland first involved, with one other, was removed for histological examination in January of 1904, whilst the patient was under the care of Dr. Scott in the Pennsylvania Hospital. During his stay there he was treated by the x-ray, with a primarily gratifying result, the glandular enlargement disappearing almost

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entirely. During the continuation of the treatment the enlargement recurred and has been persistent since that time. One gland in about the middle of the sterno-cleido-mastoid has reached the size of an unhulled walnut, and between this and the cervical, and evidently more deeply situated, there is one gland the size of a small hen's egg.

About two years ago the boy began to cough. The cough soon became paroxysmal, worse at night, and sometimes attended by vomiting. It is laryngeal in type and rarely attended by expectoration. On several occasions there has been a slight diminution of the coughing.

With the initiation of the cough he began to grow pale and lose flesh. His parents have recognized that he has been feverish at times, and more especially when his cough has been most severe. For some months past he has suffered from dyspnea on exertion. He has never been edematous, and his general health has been good. He has never suffered pain in any part of his body, nor have the glands been tender.

The physical examination showed the glands in the neck as noted above. In addition, there exists a marked fulness in the veins of the upper part of the chest, extending slightly to the right, but much more evident on the left side, and involving the left shoulder. The sub-, as well as the supera-clavicular, fossa on the left side is fuller than on the right. The left forearm and hand are larger than the right. There is more marked congestion on the left hand and forearm than on the right, and the superficial temperature is lower on this side and the volume of the pulse much lessened. There is an area of dullness over the upper parts of the chest, extending on the right almost to the midclavicular line, and downward and inward to the third costal cartilage and to the midclavicular line on the left, being bounded on the top by the upper part of the sternum. Aside from a harsh, inspiratory murmur on both sides, there are no changes in the lung. There is no effusion in the pleural cavities; the heart is not abnormal.

The spleen is very much enlarged, extending to the transverse umbilical line and to the median line on the right. The upper border of splenic dullness extends to the sixth left space in the mid-axillary region.

The liver is felt an inch below the costal margin in the extension of the midclavicular line. Its upper border of deep dullness begins at the third rib in this line, anterior axillary line at the

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fourth rib, the posterior axillary at the sixth rib and just to the right of the vertebral column in the eighth interspace.

There is no enlargement of the other superficial glands, nor evidences of enlargement of any of the abdominal glands. The boy is decidedly anemic.

A blood examination, made by Dr. Longcope on the 26th of January, 1904, when the patient was in the Pennsylvania Hospital, resulted as follows:

Erythrocytes	6,280,000
Leukocytes	10,900
Hemoglobin	90 per cent.

Differential Count.

Polymorphonuclear leukocytes	81.0	per cent.
Large mononuclears	17.5	" "
Small mononuclears	0.5	" "
Eosinophiles	0.5	" "
Transitionals	0.5	" "
	100.0	

A blood examination made the 16th of March this year at the Polyclinic Hospital, showed:

Hemoglobin	65	per cent.
Red blood corpuscles	3,700,000	
White blood corpuscles	6,400	
Polymorphonuclears	56.4	per cent.
Lymphocytes	34.0	" "
Transitionals	0.8	" "
Eosinophiles	8.8	" "

The latter finding, if proven to be correct, shows a remarkable number of eosinophiles, a condition which would prove unique and interesting in the light of the great abundance of eosinophiles which are found in the glandular enlargements as well as in the bone marrow. The examination also marks a very decided increase in the anemia since the original examination by Dr. Longcope.

During the time that this boy has been under observation,

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there has been a persistent slight elevation of temperature, the highest record being 100.5°F.

Through the courtesy of Dr. Longcope, I have been enabled to submit his report upon the microscopic examination of the glands removed, the principal features of which are the almost complete destruction of the normal structure of the gland, increase in the lymphoid elements in certain areas, the proliferation of the endothelioid cells, the presence of uninuclear and multinuclear giant cells, eosinophiles in great profusion, thickening of the reticulum and overgrowth of the connective tissue.

Dr. Longcope had been very much interested in this boy because he had been under x-ray treatment. In a number of cases he had had an opportunity of studying the glands both before and after treatment with the x-ray, especially in a fatal case in which all the glands were examined. As the glands became smaller there was a distinct degeneration of the cellular elements. There was an overgrowth of connective tissue with a hyaline degeneration, only a few islands of cellular tissue remaining. This is the principal effect of the x-ray upon the glands. He doubted the possibility of securing permanent results from this form of treatment, for he had noticed that, even in patients in whom the glands were much reduced in size, if treatment were suspended for several weeks, the glands invariably became enlarged again.

Dr. Graham thought it a special privilege to be allowed to see 3 cases of Hodgkin's disease in one evening. All these cases showed marked cervical enlargement, but very little enlargement of the axillary glands. He thought that the general opinion at present is that these cases are nontubercular. He mentioned a case that he had observed about a year and a half ago, in which the cervical, axillary and abdominal glands were much enlarged. The glands were studied during life with the idea, if possible, of determining whether they were tubercular. The histologic appearances were those usually described in Hodgkin's disease, and, furthermore, guinea-pig inoculation was negative. Postmortem all the glands were carefully studied, but no evidences of tuberculosis were found. Dr. Graham considered Hodgkin's disease an entity absolutely distinct from tuberculosis, and thought that the two diseases had been confused for the reason that many cases of tuberculosis of the glands had been called Hodgkin's disease.

Dr. Hamill asked Dr. Longcope if it was likely that as high a

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percentage of eosinophiles should occur as was indicated in the blood examination of one of the cases shown. He said that he was aware of the fact that the percentage of eosinophiles in the blood was ordinarily extremely low, whilst they were present in great profusion in the glands.

DR. LONGCOPE, in answer to Dr. Hamill's question, said that it is very unusual to have an eosinophilia associated with Hodgkin's disease. In fact, he was not aware that any case had been reported in which the percentage of eosinophiles was increased in the blood. It would, however, not be surprising if such a condition were found, since, in the lymph nodes these cells so often occur in great numbers.

DR. MILLER said that he had forgotten to mention the most interesting feature of his case, viz., the treatment. The boy had been under treatment with the x-ray for six weeks, but there had been no appreciable improvement in the condition.

DR. HAMILL showed a case of

AMAUROTIC FAMILY IDIOCY.

The patient was a female child of Jewish parentage, aged twenty-two months. Her parents are living and well, and they were not related before marriage. The only other child born to them died at the age of thirteen months in a convulsive seizure, occurring in the course of an illness of two days' duration. Prior to this, however, it was known to have had "stomach trouble."

A maternal uncle, aged twenty years, had had a nervous disorder, which had incapacitated him for walking for the past six years.

The child had had no illness other than the present. It was breast-fed for thirteen months, cow's milk being added to the diet at the age of seven months. Since the end of the first year she had received, additionally, eggs, and, occasionally, tea.

Present History.—The child was perfectly well until six months old. She then became very constipated, vomiting occasionally, lost some flesh, and grew gradually weaker. At the beginning of this trouble it was noted that she was unable to hold her head up as well as she had formerly done. She has never been able to sit alone, but she came nearer to it prior to the sixth month than since then. Until she was one year old, she was able to see and would pick up objects placed near her. Since then she has

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ceased to notice objects, which her parents have attributed to defective mental development. It has also been noted that, coincident with this indifference to objects placed about her, her intelligence decreased, and the arms and legs, which had been formerly more or less flaccid, became spastic. The mother states that, recently, she has had attacks of extreme rigidity, which persisted unaltered for periods of one hour. The spasticity is not constant; there is frequently a momentary relaxation of one or two members. Sometimes the rigidity occurs so suddenly that the leg or arm is thrown into a state of spasm. The mother has never noticed any involvement of the muscles of the face, neck and trunk. She has attacks of laughing, which come on with marked suddenness and without cause, sometimes continuing for ten or fifteen minutes ("explosive laughter"). She has always had some cough, which has been worse the past few days, and seems to be the result of a tendency to choke. She has some difficulty in swallowing, and recently has refused all food except milk.

There is some redness and swelling of the gums, a moderate amount of redness of all of the mucous membranes of the mouth and marked salivation; no bleeding. She is supposed to be feverish quite frequently. Her bowels are persistently constipated, being moved with difficulty ever since the illness began. Her hearing is good and she is very much startled by sudden sounds.

The main features at the examination are a peculiar blank expression, dribbling of saliva, falling forward of the head, spasticity of the arms and legs, which can be readily temporarily overcome by resistance; some rigidity of the muscles of the back of the neck, marked increase in the knee jerk and the plantar reflex, marked hyperesthesia on the arms, and especially the legs, persistent extreme extension of the great toes and flexion of the small toes, a peculiarly shaped head—rather narrow anteriorly, broad in the biparietal diameter, flat in the occipital region, producing a triangular shaped cranium and an anterior fontanel, which is about 1x1 inch in diameter.

Dr. Posey, by whom the eye examination was made, reported that the typical lesion of amaurotic family idiocy was present. His detailed report had not been submitted.

Dr. Hamill said that this condition is evidently becoming more commonly recognized than formerly. Within the past two years, he had had 3 cases under his care and had knowledge of several others.

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This increased frequency makes them less interesting from a clinical standpoint, whilst the difficulty in securing autopsies, on account of the race prejudice, continues our interest in their pathology.

The widespread degeneration of the ganglion cells of the brain and cord, first observed by Hirsh, have been confirmed by Sachs and Spiller. The same lesions have been described by Spiller and others in other conditions. It is questionable, therefore, whether these findings should be considered characteristic. There is probably some other factor, as yet unrecognized, which must be developed in order to account for this distinctive symptom complex.

TWO CASES EXHIBITING NERVOUS SEQUELÆ OF SCARLET FEVER.

DR. MILLICENT HOPKINS presented these patients. The first was a boy of four years, of Italian parents. The father and mother are living and in good health. There are four other children, all well; one died at ten months of hemorrhage. This child was well until two years old, when he contracted scarlet fever. During the course of the disease he had a very severe convulsion, and later the mother noticed a paralysis of the right leg, right arm and right side of the face.

Examination by Dr. Spiller was as follows: The right lower limb is flaccid and smaller than the left; there is no ankle clonus; the right patellar reflex is lost, while on the left side it is increased; there is no Babinski reflex on either side. The gluteal fold of the right side is obliterated; sensation is normal. The upper limbs are well developed, the right being a little smaller than the left. There is no distinct facial palsy. There is no response to the faradic current from the anterior tibial muscle, and but slight contraction of the long extensor, on the right side.

Diagnosis.—Anterior poliomyelitis, probably with some encephalitis, the result of scarlet fever.

The second patient was a girl of fifteen years, born in Russia. Both parents are living and well. This girl had always been in good health until an attack of scarlet fever at seven years of age. During the illness a convulsion occurred which left a right hemiplegia. The mother tells particularly of the slow return of speech. There is spasticity of the right arm and leg, more marked in the arm. The patellar reflex of the right side is diminished. There is a slight Babinski reflex on the right side. There is

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wasting with marked contracture of the right arm, and this member can be raised only to a right angle with the body. There is mental impairment.

Diagnosis.—Hemiplegia, either from hemorrhage or encephalitis, the result of scarlet fever.

DR. GRIFFITH said that within a year he had had occasion to look up some of the journal literature pertaining to scarlet fever, and that he had been impressed with the scantiness of reference to nervous lesions accompanying or following the disease. The 2 cases shown this evening possessed, therefore, a special interest. They were certainly very unusual.

DR. MCKEE thought that the cord case was most remarkable. He had consulted several authorities upon the subject, but could find no references to such cord lesions complicating scarlet fever.

DR. MILLER thought that cases of this sort must be very rare, He remembered in reading Meigs and Pepper's "Diseases of Children," the report of a case of general paralysis developing during scarlet fever. The patient was about a year recovering. The condition was probably a multiple neuritis; although the authors did not so state. This was the only instance of paralysis following scarlet fever that they had seen in their enormous experience.

DR. EDWIN E. GRAHAM reported a

CASE OF RHEUMATIC ENDOCARDITIS ENDING FATALLY.

The full report of this case will be found in this volume, among the "Papers," page 36.

DR. MILLER asked Dr. Graham what he considered the cause of death in this case, and. also, whether there had been tonsilitis preceding the rheumatic attack. He thought that in many cases of rheumatism and endocarditis the infection was introduced through the tonsils. He, also, was inclined to question Dr. Graham's conception of his case as one of simple endocarditis. The child was seriously ill, and the case ended fatally, after a brief illness; events of infrequent occurrence in simple endocarditis. Without blood examinations during life, and microscopical and bacteriological examinations after death, it was not possible to say that any given case belonged to the simple or the socalled ulcerative (infective) variety of endocarditis, especially when a case terminated fatally, as this one had done.

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DR. ROUSSEL thought that occasionally in rheumatism the heart manifestations precede the articular symptoms. He mentioned the case of a child of six years, who, several days after an ordinary attack of tonsilitis, developed endocarditis. Subsequently, the articular symptoms of rheumatism were observed. He quoted the late Dr. Packard's writings upon tonsilitis as the source of infection in endocarditis.

DR. MCKEE mentioned a case which first came under observation with a follicular tonsilitis. This was followed several days later by an erythema multiforme, fever and signs of an endocarditis. Under treatment with the salicylates, the temperature became normal, and the eruption disappeared. The patient finally developed pericarditis and myocarditis and died.

DR. GRAHAM said that there had been no tonsilitis in this case, and no history of any infectious disease. He felt confident that it was a case of simple endocarditis, as there had been no chills or septic temperature. Death was very sudden. The child awoke terrified from a dream and died within a few minutes.

A BRIEF NOTE CONCERNING THE EXPRESSION OF THE MILK FORMULA.

DR. J. H. MCKEE spoke of mathematical formulæ as representing a universal language. The more graphic a formula is, the more clearly it speaks. He contended that milk formula might be made more graphic. Instead of expressing a formula—fat, 3 per cent.; milk sugar, 6 per cent., and proteid, 1 per cent.—it would be better to say: Proteid, 1 per cent.; fat, 3 per cent., and milk sugar, 6 per cent. The latter order possesses the following advantages over the former: (1) It follows physiologic custom; as in the expression of the "normal" or "average diet" of the adult. (2) It expresses these organic constituents in the order of their metabolic values. (3) It expresses the order in which these groups are liable to cause digestive disturbances. (4) It expresses their quantitative ratios, one to another, in natural sequence.

DR. GRAHAM thought that if an effort were made, Dr. McKee's suggestion would probably be taken up. Of course, it would be difficult to depart from a method which custom has made familiar.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, May 9, 1905.

JAMES H. MCKEE, M.D., PRESIDENT.

SCLEREMA NEONATORUM.

DR. J. P. CROZER GRIFFITH exhibited the infant, aged ten weeks, which was recovering. There was no discussion.

CEREBROSPINAL MENINGITIS.

DR. ALFRED HAND, JR., who was to have shown several patients with meningitis, regretted his inability to do so because one had died, one who had recovered, showing no trace of cerebrospinal fever, had gone home, and the three remaining in the hospital were all too ill to be brought; one had tuberculous meningitis, and the others were instances of epidemic cerebrospinal meningitis. Dr. Hand wished to demonstrate under the microscope a slide from the case of tuberculous meningitis, showing tubercle bacilli and a predominance of lymphocytes, and also a slide from some fluid sent him by the President, Dr. McKee, showing the diplococcus intracellularis with an abundance of polynuclear leukocytes. The chemical examination of the fluids from these 2 cases was strongly indicative of what the bacteriologic result would show, that from the tuberculous meningitis showing a large amount of albumin with the presence of sugar, while that from the epidemic meningitis showed albumin moderately increased and sugar totally absent; the latter condition also holds for pneumococcic meningitis, but when the former is present, increased albumin with sugar, meningitis being suspected, it is strongly diagnostic of tuberculous meningitis. "At least, in all such cases that I have examined," said Dr. Hand, "it has been possible to put on the capstone by demonstrating the tubercle bacillus."

DR. GRIFFITH referred to the case which he had exhibited to the Society three months before, and asked whether Dr. Hand could tell its further history. The patient, who was then in the one hundredth day of illness, afterwards grew worse, while still under his care, with a tendency to return of rigidity and hyperesthesia.

DR. HAND replied that that boy was one of the patients whom he had intended to show on this occasion; but that during the

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last six days the patient had had two attacks of fever without any lesion to account for it. This might be explained as a fresh invasion of the meningococcus. Having just finished one of these attacks, the patient's mind was as clear, apparently, as it had been. He could answer questions, was not wasting any, and showed about the same condition as when exhibited. This is the most protracted case of the disease that Dr. Hand has ever seen, as the boy has now been sick for one hundred and eighty days. A few years ago there occurred, in Dr. Hand's service at the Children's Hospital, a case which lasted one hundred and ten days, a curious feature being ephemeral attacks of hyperpyrexia, the temperature rising from its usual range of about 100° to 106° or 107° F. in a few hours, and then subsiding as quickly as it rose; these came at irregular intervals of from five to ten days.

Dr. McKee said that he should like to refer to the case mentioned by Dr. Hand, for it showed the extreme value of lumbar puncture as an aid in diagnosis. Dr. McKee saw the patient on the eleventh day of illness, and again on the twenty-third day; and he had been informed by two physicians, who had seen him previously, that it was probably syphilitic meningitis. In addition to unquestioned meningitis, the boy presented a typhoid facies, malar flush, typical typhoid tongue, sordes on the lips and gums, a moderate degree of hypostatic congestion, weak first sound, slow pulse, enlarged liver and enlarged spleen; and even in his semiconscious periods, he was tender in the right iliac fossa, and there was a great deal of gurgling present. In spite of the history, which was of sudden onset, accompanied with vomiting and high temperature, Dr. McKee was inclined to consider the case not meningitic typhoid, but a true typhoidal infection of the cerebrospinal meninges. He had seen one such case in which the history and examination were more misleading. On the twenty-third day a lumbar puncture withdrew 50 cu. cm. of fluid, which Dr. Hand had examined the day before the meeting. It was the most turbid fluid that Dr. McKee had ever seen; and he had never observed fluid spurt so far from the end of the needle. When the boy cried, the fluid spurted at least three inches.

Dr. McKee read Dr. Hand's report of the examination of this fluid. He also said that from the therapeutic standpoint he desired to add that the boy had partially regained consciousness before he (Dr. McKee) had left the house, after having been either unconscious or semiconscious for three weeks. His tem-

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perature fell from 104°F. on Sunday afternoon, when the puncture was made, to 101.2°F. on Monday morning, and had fallen progressively since then. That afternoon it was a little over 99°F. The case is one of those in which a marked, even though temporary, therapeutic result followed lumbar puncture.

In answer to Dr. John H. W. Rhein's question, whether he had ever found the staphylococcus pyogenes aureus in cerebrospinal fluid, Dr. Hand said that he had often seen in cerebrospinal fluid cocci which he had looked upon as a contamination. The slide with the tubercle bacillus, which Dr. Hand exhibited, showed also a diplococcus, not like either the pneumococcus or the diplococcus intracellularis, and not bearing any relation to the cells. It had appeared a day after the withdrawal of the fluid, and had made it cloudy. Dr. Hand thinks it essential to take the precaution of singeing the cotton stopper of the test-tube after it had been held in the air, but this was not done in this case; and to this omission Dr. Hand attributes the presence of the diplococcus. He has always looked upon the presence of other germs than the pneumococcus, the diplococcus intracellularis, the tubercle bacillus and the typhoid baciilus in such cases as most likely to be the result of contamination.

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THE INHIBITORY ACTION OF UNHEATED MILK UPON BACTERIA.

Drs. J. S. Evans and T. A. Cope read a paper with this title. Different investigators, who have taken up this subject since the discovery of the bactericidal property of blood serum, have based their conclusions either for or against the presence of this property in milk upon experiments with milk which has not been absolutely sterile; on this account results have been varied. It has been the experience, however, of all workers that in heated milk a more rapid multiplication of bacteria occurred than in the natural product. This fact is taken by many as a proof that the heating of milk destroyed some substances similar to that found in blood which had the power of either inhibiting or destroying germ life.

The present experiments were conducted in order to determine whether this apparent bactericidal action was due to the property of the milk or due to the antagonism of one species of microorganism to another. To accomplish this it was necessary to obtain sterile milk. This was done by a specially devised apparatus and the experiments were conducted similar to those in testing blood serum.

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The natural product, milk heated to 55°F for thirty minutes, 68°F. for twenty minutes, 100°F. for thirty minutes, and frozen, were used as a basis of the experiment.

A series of these specimens were inoculated; one with the streptococcus, one with the staphylococcus, one with the bacillus coli communis, one with the bacillus subtilis, one with the bacillus acidi lactici and one with a mixture of the two latter. Counts were then made immediately after inoculation, and four, eight and twenty-four hours later. It was found in each of the series that the percentage of increase was greatest in the sterilized specimens, next in the pasteurized specimens, and next in the specimens heated to 55°F. In the specimens inoculated with the mixed cultures it was found that the bacillus acidi lactici rapidly killed off the bacillus subtilis as well in the natural specimens as in the heated ones, which fact gave some proof for the belief that the results obtained by other investigators were in a measure influenced by bacterial antagonism.

The physical changes, that is, coagulation and digestion of the curd, went hand in hand with the character of the organism and the increase of its multiplication.

Conclusions to be drawn from these experiments are:—

(1) Unheated milk does possess a slight inhibitory action to the varieties of organisms used. This action at incubator temperature lasts from four to eight hours.

(2) Physical changes in milk have a direct relation to the number and variety of bacteria present.

(3) The antagonism of one species of microorganisms toward another must be considered in drawing conclusions from experiments made with milk containing the mixed bacterial flora.

Dr. Griffith said that, before the regular discussion was opened, he should like to know whether the curves on the charts shown represented the actual numbers of bacteria or only the rate of multiplication.

Dr. Evans said that they represented the latter.

Dr. Griffith replied that these showed, then, that, although pasteurization did not diminish the rate of multiplication, it did exert a favorable action in vastly lessening the actual number of germs.

Dr. Evans said that the question had come up in reference to commercial milk. In making tests of this, a count of 100,000

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would have a mixed bacterial content. During pasteurization, the bacteria not killed would be the spore-forming organisms. The pasteurized specimens were found to contain fewer bacteria at the end of forty-eight hours than the original milk. The percentage of increase, however, he said, was greater.

Dr. M. P. Ravenel opened the discussion by saying that he had followed this work with a great deal of interest. The subject had been first called to his attention by some work done by Dr. Shaw in the laboratory of the State Live-Stock Sanitary Board, in 1897. After watching this work for some time Dr. Ravenel had come to the conclusion that a bactericidal action in milk had been demonstrated. The results of this work have never been published. Dr. Ravenel afterwards found that several observers, among whom was Dr. Park, of New York, had succeeded in demonstrating the same power in fresh milk. He said that it has been conclusively shown that immune-bodies may pass into the milk of immunized cows; so that the bactericidal power of the blood could pass, to some extent, into the milk.

Dr. Ravenel then remarked that, while he considered the work of Drs. Evans and Cope as the crown of all the work done along this line, he did not think that, practically, it tells us what we want to know. That pasteurized and sterilized milk will coagulate more quickly than fresh milk, under the same conditions, has been shown before. The practical question is whether, in handling the milk and putting it on the market as a commercial product, it should be pasteurized or not. It has been absolutely demonstrated by the work seen here that the pasteurization of very pure milk is folly. A considerable part of the work done by the Philadelphia Pasteurizing Society is gratuitous. To take a very pure milk and pasteurize it is wrong; for pasteurization produces, in certain conditions, malnutrition, while the keeping quality of the milk is not improved.

The mistake that the speaker considered to have been made in the work of Drs. Evans and Cope was that their tests had not been made with mixed milk. A very complete piece of work along this line had been done by Mr. Hunziker, of Cornell, who had tested the milk of fifteen cows, both individually and in mixture, at temperatures of 40°, 55°, 70° F., and even higher. He found that the bactericidal power varied tremendously with different cows. In some the bactericidal action would reduce the number of bacteria to a seventh of the original number. The

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most marked bactericidal power was observed at 70°F., but was always of shorter duration than at lower temperature. It was always at an end by the twelfth hour, with an average duration of three to six hours, while at lower temperature (under 50°F.) it persisted twenty-four to thirty-six hours.

Mr. Hunziker noticed another point, slightly touched upon by Dr. Evans. He found that the acidity of the milk did not increase in proportion to the increase of bacteria, in a great many instances. In a large proportion of the samples of milk the acidity did not begin to increase until fifteen or twenty hours before coagulation; and at the time it did start to do so, the bacteria were innumerable.

For ordinary practical purposes Dr. Ravenel is a firm believer in the use of pasteurized milk, but he prefers certified milk when it can be obtained. Owing to the expense of the latter it does not seem that it will ever be possible for the community in general to use it. In the absence of certification, pasteurization must be resorted to. He, therefore, desired to know whether the more rapid curdling of pasteurized milk contraindicates its use. His own idea is that in the majority of cases it would do so; but he said that it should be remembered that many bacteria secrete a rennet-like ferment which produced a more or less sweet curd. As shown at the meeting of the Pathological Society, February 23, 1905, these milks do not show an increased acid-production, although they curdle more rapidly. The result is that there is formed a curd that is probably digestible and harmless. While not wishing to be considered as an advocate of the giving of curdled milk to infants, Dr. Ravenel thought that pasteurized milk on its way to the curdling stage would not do much harm.

The value of pasteurized milk has been demonstrated clinically the world over in innumerable instances. It would do great harm to inculcate a prejudice against its use generally. The charity of Mr. Straus in New York is ample demonstration of its value. The other advantage possessed by pasteurization is the complete elimination of all pathogenic bacteria. This has been absolutely proved. If, as Dr. Evans had suggested, the pasteurization be done in the bottle in which the milk is to be delivered, no reinfection can occur.

Dr. Ravenel thinks that further study as to the reason of the curdling of sterilized and pasteurized milk should be made, and that further effort should be made to learn the nature of

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the substance that causes the coagulation, whether a ferment or an acid. He felt sure that in many cases the substance is a ferment, and not an acid. In addition, he suggested that the work should be repeated at different temperatures, it having already been performed at ice temperature and at incubator temperature. The germicidal property varies with the temperature at which the milk is kept. In concluding, Dr. Ravenel said that no one who has not been accustomed to doing laboratory work could appreciate the amount of labor that must have been expended by Drs. Evans and Cope in this investigation. He thought they were to be congratulated upon its accomplishment, and that the Society should be congratulated upon having the opportunity to listen to the presentation.

Dr. Griffith, in discussing the paper from the clinical side, said that it opens a subject a little wider than that to which the authors especially referred. He should, therefore, like to extend his remarks to the question of milk as a live thing. For a good many years physicians had been trying to obtain modifications of cow's milk that would make it like that of human beings; and great difficulty had attended this attempt. A mixture with apparently the same proportions could be obtained, but, when fed to the child, it did not produce the same result. It is only within comparatively few years that investigators have been laying more stress upon milk as being something alive, or, perhaps more strictly, as containing in it the remains of a living, cellular action. Upon this fact depends not only the possibility of the presence in the milk of bodies destroying bacteria, but also that of the various ferments which certainly exist in it. It is probably largely due to these ferments, as well as to other insurmountable chemical differences, that the digestibility of human milk is greater than that of the milk of cows. There is in human milk a whole series of ferments that do not exist in cow's milk, or exist only to a limited extent. One of these is the very much talked of amylolytic ferment. It is curious that this should be present, since at the period of the child's life when human milk is received, there is no expectation that starch will be ingested. This is probably the explanation of the claim made that the child fed on human milk is able to digest starch better than one that takes cow's milk. The presence of a fat-splitting ferment, a fibrin-breaking ferment, and an amylolytic ferment emphasizes the difference from cow's milk, in which they exist to a limited ex-

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tent, if at all. On the other hand, there are ferments, such as oxydase, present in cow's milk, but absent, or nearly so, from human milk. There are also differences in the caseins of milk that chemical examination does not reveal. This is proved by the experiments of Bordet who injected into one animal the milk of another, developing in the blood-serum of the injected animal a substance that could coagulate the casein of the milk of the animal whose milk had been used.

The various ferments of human milk are all destroyed by heat, and this is probably true of the bactericidal bodies called alexins. Certainly the experience of Drs. Evans and Cope indicates that heat decreases the bactericidal properties of cow's milk. All these considerations support scientifically the commonly accepted view that breast-milk forms the best food for the infant; but that, when this cannot be had, cow's milk, in a natural, live state, is the next best thing, no foreign action, such as heat, having been employed. It is for the obtaining of this sort of milk that the Society has been working for a number of years; and it had been hoped that when the public interest, in the newspapers and on the part of the Board of Health, was aroused, it would have been continued along the line of trying to procure clean, natural milk, instead of trying to remedy by pasteurization or sterilization milk that is not clean, and that cannot be made so. The bacteria may all be destroyed by heat, but the poisons already produced through bactericidal action will not be necessarily destroyed. When clean milk can be obtained, there is no object in heating it, and decided objections to doing so. It has long been known to clinicians that pasteurized milk requires greater care in its keeping than does raw milk. Dr. Griffith emphasized the fact that nothing he had said militated against the use of pasteurized milk under certain conditions, and prepared in a proper way. Pasteurizing is, however, an exact process, and must be done carefully. He said that he does not trust the commercially pasteurized milk at all, never prescribing it, and advising against it, when he finds that it is already in use. If one advises parents to buy pasteurized milk, one is giving them a false sense of security. The mere fact that a milk wagon has the label "Pasteurized Milk" upon it does not make the milk good. The parents, however, feel that it does. Much harm is done in this way. The same objection holds against a large proportion of the milk which is advertised by the dealers as "Certified." Dr.

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Griffith much prefers to have parents get the best and cleanest milk that they possibly can. This should be certified to in a proper manner, as the Pediatric Society is doing. Persons who do not live in the city, and those in the city whose means do not warrant it, cannot obtain this certified milk, and have to get the best they can. Under these circumstances, Dr. Griffith would rather have them use pasteurized milk than anything else, but the pasteurization should be done at home in the proper manner, and the proper care be taken of the milk afterwards. Even if the bactericidal action of the milk is lessened by pasteurization, this is better than not to insure the destruction by heat of most of the bacteria in other milk.

Dr. Mary E. Pennington said that the commercial side of the question was the one that she had seen the most of. She heartily agreed with Dr. Griffith in regard to that, for she thought that after pasteurization milk is often richer in organisms than before. This is not because pasteurization does not remove them, but is due to the fact that in the process of getting the milk into the bottles more organisms are picked up than were in the milk in the beginning, as indicated in counts made by her.

Dr. Pennington thinks that the question of modifying milk for children should be considered in this connection; for when cream is added, together with milk sugar, to even a clean milk, there is afforded a means for the entrance of numerous micro-organisms. Cream almost invariably contains more organisms than does milk, and the commercial milk sugar is not bacteriologically clean. Therefore, something should be done, if it is desired to have the milk as fed to the child clean. Dr. Pennington does not see why this could not be readily accomplished by sterilizing the mixture of cream, milk sugar and water before adding it to milk. The proteid in the cream is in but small amount, and the fat would not ordinarily be injured by the short boiling required. By this means the number of organisms could be reduced to almost none at all, if the milk to which the mixture is added is itself clean. Such a mixture will keep perfectly well for more than twenty-four hours.

If, as is the usual custom, lime-water is added, there will be no increase, but often a decrease at the end of twenty-four hours; and Dr. Pennington believes pasteurization to be unnecessary in that regard. She has recently been making some experiments that are rather interesting from the standpoint of Dr. Evans'

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paper; testing some clean milk removed from the can with a dipper sterilized in the usual hospital fashion, put into a clean vessel, covered with a sterilized towel and carried through corridors and up the stairs to a room in which it is bottled and modified. The samples, when first taken from the can, contained between 3,000 and 4,000 bacteria. After the milk had been taken upstairs, the number had increased to 14,000. The number of bacteria in the samples of clean milk had increased but slightly by the following day; the number in the milk taken upstairs, which was kept in the refrigerator, had decreased by the following day. This experiment has had the same result so many times that Dr. Pennington considers it established that the clean milk has some inhibitory action upon the dust germs that fall into the milk while it is being carried about.

Dr. T. S. Westcott said that Dr. Pennington had suggested a very important point in the management of cases of infant feeding. He has always held that cream is a fertile source of trouble in disturbances of digestion due to milk, especially in the poisoning cases. Unless the cream is produced by a centrifugal process, it is naturally older than the milk with which it is mixed; therefore, its bacterial content is usually higher also. Cases of milk-poisoning should often, and possibly in most cases, be called cases of cream-poisoning. Dr. Westcott thought that in summer the danger from cream is greater than from milk. For this reason it seemed to him that Dr. Pennington's suggestion that the small portion of cream should be boiled for a short time before being added to the milk is an excellent one. The sugar of milk and water can be sterilized by boiling at the same time, thus purifying the other elements of the mixture that are dangerous. In the summer Dr. Westcott is always very particular as to the source of the cream used in the milk mixtures under his control. If a centrifugal cream cannot be obtained, he directs that the cream should be obtained from milk of the same age as that used in the mixture, either by Chapin's or Townsend's method.

Dr. Westcott said he was glad that Dr. Griffith had referred to the work of the Milk Commission of the Pediatric Society. It has been a matter of regret that certified milk has not obtained the support of the profession in Philadelphia that it deserved. Several firms that were under contract with the Commission to produce certified milk have recently ceased to do so on account of the unsatisfactory monetary returns received by them. In

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fact, there are now fewer firms producing the certified milk than there were a year or two ago; though it is a fact that the firms still producing it are doing an increasing business. Dr. Westcott said that it is unfortunate that physicians do not sufficiently appreciate the value of a clean milk. To the physician who works among children a clean milk is as important as a sharp knife is to the surgeon. The tools must be good; and unless one works with good tools one cannot produce the best results. For this reason Dr. Westcott urged the members of the Society to endeavor this summer to make a more extensive use of certified milk. The cost of it is greater than that of ordinary milk, but only slightly so. When one comes to count up the cost to the parents of even a very short illness in a child, it is easy to see that the dearest milk is the cheapest in the end.

Dr. Hand said that when Dr. Ravenel had spoken of the impossibility of supplying the community with certified milk, he had thought of referring to a paper that he then had in his pocket; and that when Dr. Westcott had made his closing remarks he (Dr. Hand) became more resolved to do so. This paper, the bill of the printer for this month's certificates for the Milk Commission, shows that, while there are fewer dealers supplying certified milk than there formerly were, there is a much larger amount of the milk supplied. The gain in the amount of certified milk over that of four years ago is about double. That day Dr. Hand had had delivered to the four dairies, through the three distributing points, the certificates for the ensuing month; and he was surprised to see that the total number of certificates was 113,500, making about 300 quarts a day to Philadelphia. That amount, of course, is practically merely a drop in the bucket; but it is a matter of encouragement to those interested in clean milk to continue their missionary work.

Dr. Ravenel said that the question of popularizing the use of certified milk is one of education. With all due respect to Philadelphia, said he, there is too much conservatism when you try to start a new thing. One of the professors of the University, when advised to use certified milk, replied that he obtained milk from the same man he had been dealing with for a long time, and intended to stick to him. This dealer was found to be what is called a "platform buyer," and did not even know where the milk he bought came from. Another prominent practitioner said he did not like certified milk because it had no flavor. There have,

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unfortunately, been attacks upon certified milk even by members of this Society, who do not seem to understand that there are limits to the certification. The milk cannot be examined every day, owing to the expense. The public does not realize how often certified milk is condemned by the Commission, when it is found to be below the standard. Even the milks that are certified, month after month, are often turned down on single examinations and the dairy required to correct the faults found before the certificate is issued. If an outside examiner happens to get a specimen of this inferior milk, he will naturally say it is below standard; and he may get hold of an old bottle. Only last month the cream from one of the best dairies averaged from 5,000 to 6,000 bacteria per cu. cm.; whereas, the milk at one examination was over 300,000. Dr. Ravenel did not know how to account for it, but every once in a while this happens in the milk from the best dairies. In such cases the milk is turned down and a re-examination required before the Society's certificate is given. It is not commercially possible to have a daily examination; and even if this were done, it would be forty-eight hours before the result of the examination could become known. This would be too late to avoid the danger from the use of any particular milk to a child, as it would have been already consumed. In the same way, the chemist cannot complete his examination in time to stop the use of the milk he finds below standard. Therefore, the best the Society can do is to exercise constant supervision over the dairies. If in a sample of milk there is found, for instance, the diplococcus of pneumonia, that milk is rejected without further counting; but, as a rule, from a numerical standpoint, the milk cannot be condemned until the lot from which the sample was taken has been consumed. These circumstances should be clearly understood, and Dr. Ravenel thinks that the Pediatric Society could do good work in making physicians understand them. He remarked that he could say, without fear of contradiction, that the Commission has three or four dairies producing milk that is unexcelled in the whole world. If this milk is not good, said he, milk should be given up as an article of food.

Dr. D. J. Milton Miller said that he should like to call attention to something that has a practical bearing upon Dr. Evans' statement concerning the growth of bacteria in pasteurized milk. Dr. Miller spends his summers at a place where good milk cannot be procured. It is brought fifteen or twenty miles

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over a rough road, and cream cannot be obtained at all in summer; it sours and becomes unfit for use. At first, it was his practice to recommend pasteurization. He induced people to get the cream and pasteurize it in the ordinary way. Notwithstanding this he found that a great many children developed diarrhea. He then made it his custom to instruct the persons that consulted him to heat the milk to a much higher temperature; to put it in a sauce-pan over a quick fire, and heat it, with stirring, for ten or fifteen minutes, but not to boil it, *i.e.*, to raise it to 185° or 190° F. For a great many years he has noticed that those who employ this process have much less diarrhea among their infants than do those who do not treat the milk in this way. This fact, Dr. Miller thinks, has a practical bearing upon the subject of the growth of bacteria; it seems to show that when milk is of questionable character, pasteurization is insufficient, and it must be raised to a higher temperature. He then referred to another point which had already been touched upon—that the worst offenders in this respect are often physicians. He knows of a number of instances in which fairly intelligent doctors have insisted upon using ordinary milk in their practice. He knows a prominent surgeon who brought up his child on a really inferior milk because it had been used in the family ever since he was a boy, and by his mother before him. The child was attended by a prominent pediatrist, but it is worthy of note that it was sick during most of its first year. Dr. Miller has had under his care this winter the child of a physician who told him that the child was using certified milk. When the infant got sick, Dr. Miller investigated and found that it was really getting "Abbott's best milk," at ten cents a quart. This physician was also a teacher in one of our Philadelphia medical colleges.

Dr. Evans, in closing the discussion, said that, in regard to Dr. Griffith's remarks concerning the natural ferments, he had a specimen of about eight ounces of milk a month old, from which they have taken a culture and found it to be sterile. At the end of two weeks a macroscopic change was seen to be taking place. It was difficult to emulsify the cream; whereas, in the heated specimens, it was much more easily mixed. Dr. Evans thoroughly believes that a good many of the changes taking place are due to a natural ferment or ferments, which may or may not have an effect upon the bacteria.

With the increase of certain forms of bacteria, there is an

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increase in the acidity. Normal sterile milk is acid. When seeded with lactic acid bacilli, it was found that at the end of twenty-four hours there was an increase of from three to four points of acidity. This was more marked in the heated specimens, which in turn had a greater number of bacteria; whereas, in the subtilis there was a diminution of .1. This point was not mentioned in the paper, because the authors deem it necessary to perform the experiment a number of times, so as to be sure of the result.

ENCEPHALITIS AND OTHER NERVOUS AFFECTIONS COMPLICATING SCARLATINA.

Dr. John H. W. Rhein reported a case of scarlet fever in a girl of nine years, who presented the usual manifestations of the disease, together with a tremor of the general musculature, which appeared a few hours before death. The microscopic study of the brain revealed the presence of a mild inflammatory change in the cortex, as well as a moderate degree of meningitis. He reviewed the literature of the subject, and was able to conclude as follows:—The most common complications of scarlatina, on the part of the nervous system, are hemiplegia and peripheral neuritis. More rarely this disease may be followed by paraplegia, optic neuritis, amaurosis, tetany, pseudoataxia, neuralgia, epilepsy, disseminated sclerosis, Friedreich's ataxia, hysteria, hydrocephalus, meningitis and disordered mental states. Some of these conditions, like Friedreich's ataxia, are probably merely hastened in their manifestations by the scarlatina. With the exception of hemiplegia and imbecility the prognosis is good, if we exclude those rare organic cases, such as Friedreich's ataxia, epilepsy and disseminated sclerosis, which are recorded as following scarlatina. The pathologic findings consist of thrombosis, embolism, small cerebral hemorrhages, rarely abscess of the brain, congestion of the brain and meninges, and, finally, meningitis and encephalitis.

Dr. B. F. Royer said that he did not feel at all competent to discuss the pathological histology of changes in the brain and meninges, which Dr. Rhein had presented so admirably. He thought that in the particular case reported, two points should be considered before drawing conclusions. As pointed out from the clinical standpoint, the child was convalescent from measles, the rash of which was fading at the time of admission to the hospital; and some of the changes noted might have been due to

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the other exanthem. The patient also had a severe nephritis and a fibrinous pericarditis. It was, therefore, a septic case, and not scarlatina alone. Dr. Royer went on to say that, if he recalled the case correctly, there was also suppurative otitis media. On admission, the child had a marked exudate on both tonsils, dark, brownish-gray, and very profuse. Cultures made from this exudate, from the surface and beneath, did not show Klebs-Löffler bacilli, but did show streptococci. Thinking that there might be some diphtheritic element, antitoxin was given. After a few days her pulse became weak, and adrenalin chlorid was then administered. The reaction from this preparation was more marked than was desired. The patient developed extremely high arterial tension, and some of the perivascular changes might have been due to this overstimulation.

The findings are very interesting. Most of the quotations given by Dr. Rhein are from other countries than the United States. This is because at the present time scarlatina in this country is of a mild type, and nervous manifestations are not prominent. In Germany, the death-rate from scarlatina is running from 12 to 15 per cent., while in this country it runs from about 6 to 7 per cent. In England it is as low as 3 per cent. At the present time physicians are not seeing the number of nervous symptoms that were formerly rather frequently observed. The case quoted by Dr. Rhein showed not only intense angina, but the rash itself was of that intensely severe type in which there are bluish-red petechiæ. The usual circulation changes were absent. It was with difficulty that the blood could be pressed out of the superficial areas of the skin, and both legs and arms had distinct hemorrhagic petechiæ. This constituted an unusual type of scarlet fever, not the type usually seen in the United States. Dr. Royer concluded by remarking that he was very glad to have heard the paper and to have had the pleasure of discussing it.

Dr. Griffith said that he wished to express his appreciation of Dr. Rhein's valuable paper, which he hoped to see published. He considered it to be a valuable resumé of the subject. His own knowledge of such conditions in scarlet fever is, he said, slight, for the reason given by Dr. Royer, viz., that the vast majority of cases of this disease are of a mild type. The literature of scarlet fever speaks of sequels or complications very rarely in the nervous sphere, as compared with their frequency in some other diseases.

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THE PHILADELPHIA PEDIATRIC SOCIETY.

Stated Meeting, Tuesday, June 13, 1905.

JAMES H. MCKEE, M.D., PRESIDENT.

CONGENITAL HEART DISEASE.

DR. EDWIN E. GRAHAM reported this case, not because congenital heart disease was such a very rare condition, but because the case presented some unusual and interesting signs. The patient, a boy, aged nine months, was the second child, the first child dying during delivery as a result of breech presentation. During the intrauterine life of this child, the mother complained only of dyspnea on exertion. Delivery was easy and not instrumental. When the baby was three weeks old, slight blueness of the lips was noticed. At the age of five months the child had bronchopneumonia which lasted six weeks. Since then cyanosis has always been present, marked in the lips and tissues surrounding nose and eyes, with slight cyanosis of the entire face, distinct cyanosis of fingers, less marked in toes; cyanosis in both hands, and a cold and clammy skin. This cyanosis becomes much more marked at times, the increase persisting from two to twenty-four hours. The cyanosis shows in a much more marked degree in the mouth and tongue than in any other portion of the body.

The child is well nourished, has two teeth, little or no dyspnea, is breast-fed, has no vomiting and no bowel disturbance, is not fretful, and is apparently in good health with the exception of the cyanosis. The apex beat is most distinct just at the nipple. A distinct thrill can be felt over the entire cardiac area; a loud, blowing systolic murmur is heard in the mitral area, transmitted to the axilla, not transmitted to the angle of the scapula. The murmur becomes less on ascending the sternum, is fainter at the aortic cartilage, and still less distinct at the pulmonary cartilage. There is accentuation of the second aortic sound. Both sides of the heart are enlarged, the right being no larger in proportion than the left.

In arriving at the diagnosis as to the probable condition, reasoning from the symptoms, and not from the physical signs, one would be led to suppose the case to be one of pulmonic stenosis, with a probable deficiency in the interventricular septum and

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possibly a patent ductus arteriosus. In the large majority of cases of congenital heart disease, however, the murmur is at the base of the heart and not at the apex, the proportion of base murmurs to apex murmurs being about three to one. The large majority of cases of congenital heart disease show more or less cyanosis, and more than one-half of the cases showing cyanosis have been proven postmortem to be cases of pulmonic stenosis. The larger number of cases of apex murmurs associated with cyanosis have been found at autopsy to be cases presenting unusual cardiac malformation, such as transposition of the great vessels, defect in the interauricular septum, or interventricular septum, associated with a patent ductus arteriosus.

Taking all the facts into consideration, it seems that the diagnosis above mentioned is probably correct, that the child has one of the unusual cardiac malformations.

Dr. McKee said that, if his memory served him, one of Dr. Holt's cases of deficient septum ventriculorum had also presented a loud systolic murmur at the apex. In Dr. Graham's patient he thought the pulmonic second sound was markedly accentuated. This might mean a patulous ductus arteriosus. He was inclined to think that this was probably an instance of the triple lesion, pulmonary stenosis, patulous ductus arteriosus and deficient septum ventriculorum. He agreed with Dr. Graham, however, as to the great difficulties in diagnosis.

Dr. Graham said that he had made the diagnosis in spite of the absence of physical signs.

Drs. C. F. Judson and G. M. Astley showed a girl who had recovered from

ULCERATIVE ENDOCARDITIS.

The full report of this case will be found in this volume, among the "Papers," page 38.

Dr. Theodore Le Boutillier spoke of a child now under his care, with a similar condition. She had endocarditis complicating articular rheumatism, and had been very ill but is now recovering. In this case no blood cultures have been made.

Dr. Astley added that the boy had knocked his knee when in swimming and had had pain in the knee before admission. He was sent into the Episcopal Hospital in the ambulance, as a sup-

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posed case of typhoid fever, with a high temperature and enlarged spleen. Arthritis developed later in the knee.

Dr. A. E. Roussel said that cases of ulcerative endocarditis were very rare, and especially so in children. He believed that the diagnosis was made positive only after cultures had been made from the blood. Dr. Judson's case seemed to be a positive one. But he reiterated the fact that cases of recovery from ulcerative endocarditis are very rare. He reported a case some years ago, in an adult, following miscarriage and septicemia. The patient had over twenty abscesses opened, and streptococci were found in the blood. He thought it difficult in children to obtain sufficient blood to find the bacteria. In his case as much as seven or nine ounces of blood were secured before bacteria were found. In children, from whom only small quantities of blood can be taken, the cocci might not be discovered.

Besides, he said that the right heart was more often involved than is generally believed. It is involved in a relatively larger proportion of cases of ulcerative endocarditis than of simple endocarditis.

Dr. A. A. Eshner said that the microorganisms that cause rheumatism also give rise to other symptoms, sometimes without involvement of the joints, such as tonsilitis, muscular and other pains, or endocarditis. He believed that there might be a "rheumatic septicemia," giving rise to a symptom-complex varying in accordance with the structures involved. Ulcerative endocarditis is to be looked upon as an aggravated form of ordinary endocarditis; therefore it may be concluded that, in a not inconsiderable number of cases, the disease may end in recovery.

INTERLOBAR EMPYEMA.

Dr. T. T. Thomas and J. H. McKee reported this case. The patient, a girl of six and one-half years, had been under Dr. McKee's care for about five years. Her history was recorded at some length, the most important point being that she had had two previous attacks of pneumonia. (Catarrhal in infancy, croupous when three years old.) On March 21, 1905, she was seized with headache, chilliness, fever and delirium, and the following day presented the classic physical signs of a lobar pneumonia, affecting the lower lobe of the left lung. The pneumonia ran a moderately severe though ordinary course. On the fifth day she had

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a pseudo-crisis, and the following day what appeared to be a true crisis. Contrary to expectations, however, she continued to run an irregular temperature, and had some sweats. On April 2d, there was apparently free fluid in the left pleural sac, and for the next four days the superior level of the same (as evidenced by physical signs) steadily ascended.

On April 7th (sixteenth day of illness) she was seen by Dr. Thomas in consultation. On this day the dulness did not appear to be movable, and operation was deferred.

Then followed a week of improvement, succeeded by a similar period, when the patient's general condition and physical signs remained unchanged. The latter had become largely localized between the anterior and posterior axillary lines, and inferior to the fourth rib.

Two days later (April 23d), slight bulging of the intercostal spaces of the above mentioned region appeared. Dr. Thomas saw her again on the following day, and the diagnosis of empyema (almost certainly interlobar) was concurred in. Exploratory puncture was made at the classic site (seventh interspace), but as was expected a dry tap resulted.

It was decided that she be prepared for operation and anesthetized on the following day, when exploratory punctures might be made until the pus was located. The first aspiration, made in the centre of dull area, secured pus. Dr. Thomas then cut down upon the sixth rib, and excised a piece of it, considerable pus escaping meanwhile. A digital exploration of the cavity found amply verified the pre-operative diagnosis. The pus pocket was drained through two drainage tubes. The patient made an uninterrupted recovery, and on June 1st (five weeks and two days after operation) the wound was closed and dry. The gain in bodily weight was quite rapid, the maximum being four pounds in a week.

Drs. J. E. Talley and J. H. Jopson reported another case of interlobar empyema, following pneumonia. The full report will be found in this volume, among the "Papers," page 43.

Dr. Graham said that empyema is commonly overlooked in children. In no disease are the physical signs so easily appreciated, yet for some reason the condition often remains unsuspected for a long time. He has occasionally found an encysted empyema, yet he never made the diagnosis of interlobar empyema.

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He has seen many cases in children under two and one-half years, and they have recovered as well without resection of a rib as after that operation. Besides, necrosis of the rib is likely to occur. He prefers incision with free drainage to resection of a rib in young children.

DR. JOPSON said that he practically always resected the rib in children, and believes it the best practice, with the possible exception of infants under eighteen months. Often the large masses of lymph can only be evacuated after resecting a rib. Yet he confessed that many young patients under his care recovered formerly, before resection was so frequently resorted to.

DR. HORACE H. JENKS read a note on

A SERIES OF CASES OF STOMATITIS.

He described the lesions in 11 cases of stomatitis which occurred recently at the Children's Hospital. Ulcers were found in the same places in each case; on the inner surface of each cheek, opposite the molar teeth, at the level of the meeting of upper and lower jaws, and below the lower incisor teeth, at the junction of gum and lip. The ulcers differed from aphthous and ulcerative stomatitis, but more closely resembled the former. They differed from the ulcerative form chiefly in the absence of involvement of the gum, in the absence of foul breath, in the absence of swollen tongue, and in their slower course; and from the aphthous form, in the small number of lesions, there being three only usually; in moderate pain; in larger ulcers being formed by extension from the original focus and not by the coalescence of smaller ones; in their yellowish color and distinct loss of tissue; in the absence of vesicles, and in the fact that they extended deeper than is usual in aphthous stomatitis. In 2 cases what were apparently Klebs-Löffler bacilli were found in the cultures made from the ulcers.

This article in full will be found among the "Papers," page 47.

DR. B. F. ROYER said he had seen a number of such cases in the Municipal Hospital. He asked whether any other children in adjoining cribs had developed true diphtheria. He had found in some cases that the organisms were not true diphtheria but pseudodiphtheria bacilli.

DR. JENKS replied that no cases of true diphtheria had developed in the wards at that time. The cultures in these cases were examined by the Philadelphia Board of Health.

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PAPERS

DELIVERED
BEFORE

The Philadelphia Pediatric Society

AND PUBLISHED IN

Archives of Pediatrics.

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I. UREMIC ONSET OF TYPHOID FEVER IN A GIRL THREE YEARS OLD; RELIEF BY LUMBAR PUNCTURE.

II. URTICARIA AS A SEQUEL OF DIPHTHERIA WHEN ANTITOXIN WAS NOT USED.

III. UNUSUAL COURSE OF MEASLES RASH.

BY ALFRED HAND, JR., M.D.,
Philadelphia.

I. UREMIC ONSET OF TYPHOID FEVER IN A GIRL THREE YEARS OLD; RELIEF BY LUMBAR PUNCTURE.

C. K., three years old, female, white, was admitted to my wards at the Children's Hospital with the history of an illness of four days' duration, the symptoms having been malaise, anorexia, fever, slight cough, vomiting and constipation, the last bowel-movement four days earlier having been very slight; she had steadily grown weaker and thinner and had been very drowsy for the past two days.

Physical examination was negative, except for a slight enlargement of the liver and some small, moist râles at the bases of the lungs; the pulse was weak and intermittent, and the general appearance was that of a very sick child, a condition which we felt was well-described by the term toxemic, and this seemed to be explained by the state of the urine, which contained albumin and an abundance of granular, epithelial and hyalin casts. A blood-count showed a leukocytosis of 12,560 and a Widal test taken at the same time, on the fifth day of the disease, was negative. When the bowels were moved, the stools contained many seat-worms.

Under symptomatic treatment, with rest in bed, there was marked improvement, so that, at the beginning of the third week in the Hospital, she was apparently in a normal condition.

On the twentieth day of the disease the temperature rose from 98.6° F., in the morning, to 103.6° F., in the evening, and was still higher on the following morning (Chart v.), when convulsions followed deep unconsciousness, the scanty urine being found to contain much albumin and many casts. The convulsive

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movements were general, but were controlled for some time by active treatment, comprising morphin, bromids and hot baths; they were returning in the afternoon, and, as there seemed to be no permanent improvement, lumbar puncture was done at 7:45 P.M., 50 c.c. of normal cerebrospinal fluid being withdrawn. The convulsions ceased and consciousness returned in about an hour. The temperature continued elevated for five days, the lowest register being 102° F., and the highest 104.8° F. At the end of this time there was a rather sudden drop one morning to 99.4° F., but the evening temperature was 100.6° F., and there then followed a febrile stadium, remittent in type and increasing in severity for ten days, when lysis began; the temperature reaching

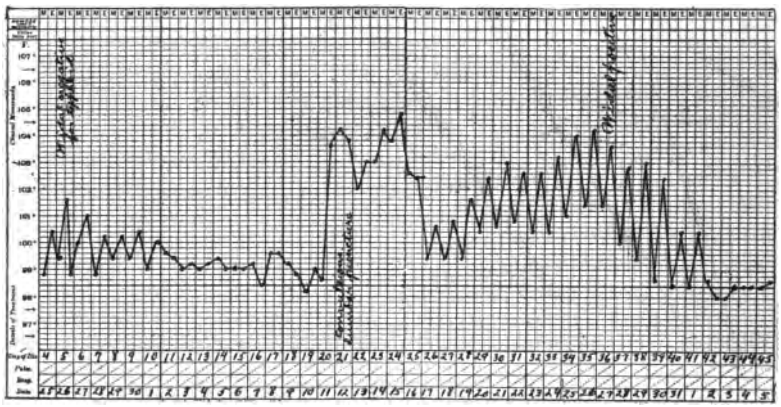

CHART SHOWING AN ATYPICAL FEBRILE COURSE, FOLLOWED BY HIGH FEVER WITH UREMIA, SUBSIDING TO A TYPICAL MILD TYPHOID FEVER.

normal twenty-two days after the convulsions and forty-two days after the original onset. Toward the middle of this last period of fever the clinical impression was that of typhoid fever, and a Widal test made on the thirty-sixth day gave a positive reaction. The subsequent course of the case was uneventful, defervescence being complete a week later and convalescence being uninterrupted.

The onset of typhoid fever with symptoms of acute nephritis occurs sufficiently often to be described in all the text-books as a type of the disease. The relief of uremic symptoms by lumbar puncture, although of comparatively recent date, has been ac-

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complished so often that the procedure is recognized as a useful addition to the treatment of the condition. The only doubtful feature of the case is the nature of the illness for the first ten days, but if, as I now believe, that was an irregular form of infection with the typhoid bacillus, then the convulsions marked the onset of a relapse.

II. URTICARIA AS A SEQUEL OF DIPHTHERIA WHEN ANTITOXIN WAS NOT USED.

There is little of importance to add to the above title except to clear myself of the charge of having treated diphtheria at the present time without antitoxin. When Frank M., seven years old, was brought to my office with a well-marked paralysis, involving the muscles of deglutition, accommodation and locomotion, I felt justified in ascribing it to an attack of tonsilitis, which he was said to have had a month earlier. The physician in attendance in that attack, the mother stated, gave no hypodermic injection, but two weeks after this the boy suffered for some days with a severe attack of urticaria, the itching from which made him restless and sleepless for several nights.

J. J. Walsh (*Philadelphia Medical Journal,* Vol. VIII., No. 15) has reported a similar case with the remark that if the conditions produced by diphtheria were more carefully studied, the diphtheria serum might be freed from many suspicions attached to it. My purpose in reporting this case is not to lay any undue emphasis upon it, but to show the possibility that occasionally an urticaria following diphtheria might have occurred even if the antitoxin had not been used. In this connection I should like to call attention to a little laxness of expression by some writers in speaking of "antitoxin-rashes." The more correct term is serum-rashes, as serum, devoid of antitoxin, may cause the eruptions.

III. UNUSUAL COURSE OF MEASLES RASH.

The unusual course in this case was the initial appearance of the rash on the legs, spreading thence to the trunk, and lastly to the neck, the face having only an erythema.

R. C., a boy of twelve years, was exposed to measles through an attack which developed in his sister shortly before Christmas, so he was kept under observation, and on the first day of the New Year he had fever of 102° F., cough and vomiting. The subsequent daily notes were as follows:

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January 2d, temperature, 100.2° F.; cough dry and hacking; January 3d, temperature, 100.4° F.; Koplik's spots present; no exanthem; January 4th, temperature, A.M., 101° F.; P.M., 103.6° F.; a typical measles rash on the thighs, faint on the trunk, none on the face or neck; face is swollen and has a sleepy expression; postcervical glands enlarged; January 5th, temperature, 103.2°-103.8° F.; the rash is most marked on the right thigh, although very pronounced on the left, the papules being very large; on the trunk the papules are small and discrete, except over the shoulders, where they are thicker with crescents; small papules on the back of the neck; face is free; flexor surfaces of arms have pronounced rash; Koplik's spots still present; January 6th, temperature, 101.4°-102.2° F.; coalescing of rash on legs, full development on chest, with diagrammatic crescents; in some places, coalescence, in some, miliaria; face diffusely flushed; tongue coated, but not the strawberry appearance; no vomiting; pulse 118; postcervical glands more enlarged; January 7th, temperature, 104.6° F.; severe diarrhea; rash has a brownish hue; January 8th, temperature, 98°-100° F.; convalescent.

The temperature chart shows a typical course with the initial catarrhal fever, the pre-eruptive remission and the eruptive rise; the subsidence of the fever was interrupted for a day by a sharp attack of diarrhea, which suggested the occurrence of an eruption on the mucous membranes.

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HEMORRHAGE OF SYPHILITIC ORIGIN IN THE NEWBORN.

BY W. REYNOLDS WILSON, M.D.,

Visiting Physician to the Philadelphia Lying-in Charity, Philadelphia.

The existence in the newborn of a tendency to leakage from the blood-vessels in the presence of abnormal conditions is a matter of common observation. The blood at this period is the most unstable of all the tissues, probably from the fact that it is undergoing the transition that marks its change from fetal to mature blood. The extravasations which are common in the neonatal period depend upon developmental conditions, as hemorrhages of this character are not manifest at birth, except as the result of trauma, and cease to be observed after the first few weeks of life. This is corroborated by the fact that prematurity and conditions of subdevelopment predispose to such hemorrhages.

Without disregarding other causes it has been demonstrated that certain hemorrhagic conditions are the accompaniment in the newborn of the more commonly recognized evidences of syphilis. A direct infection transmitted in antenatal life has been proven by the history in individual cases. Although syphilis in the fetus may so affect nutrition as to interfere with development, the form of syphilis in the newborn in which hemorrhage occurs is not necessarily accompanied by cachexia, as the infants may be well nourished. It bespeaks, therefore, a constitutional tendency.

As to the terminology, there seems to be a tendency to accept the term purpura as applicable to hemorrhagic conditions in the newborn. This designation, however, should include not only the skin hemorrhages, but internal extravasations. On the other hand, it would hardly be proper to apply to the general hemorrhagic conditions observed in the newborn the term purpura hemorrhagica which is reserved for the severe conditions seen at a later period. It appears, therefore, that the latter term should remain as a special designation, notwithstanding its applicability to the majority of instances of bleeding in the newborn.

The term syphilitic purpura is applicable to those purpuric

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conditions which are due to syphilis, but in accepting it, the tendency in syphilis, as in other conditions marked by hemorrhage in the newborn, to general bleeding, in contradistinction to skin hemorrhage, should be borne in mind.

The term hemophilia is inapplicable to the hemorrhagic conditions which enter into the differential diagnosis of syphilis in the newborn. Hemophilia is a later manifestation and bears evidences of being an hereditary tendency. It usually affects male children. This is not gainsaying the fact that persistent wound hemorrhage may occur as a complication of purpura (*vide,* A Case of Icterus Neonatorum with Purpura and Persistent Hemorrhage from a Punctured Wound, J. E. Talley, ARCHIVES OF PEDIATRICS, September, 1904, p. 695). The writer has observed one instance of oozing from the skin in a syphilitic infant. The lower lids and the flexor surfaces of the ankles were the points from which oozing took place.

The term "Hemorrhagic Disease of the Newborn," which has been adopted by Townsend and Rotch, implies a condition dependent upon a special pathological change, such as is seen in acute fatty degeneration of the newborn; whereas, the hemorrhagic tendency in the newborn is due to a variety of causes and is incidental to the development of this age. For this reason we may speak of it as a diathesis rather than a disease.

Syphilis in itself, where it can be proven to exist, is in all probability not the primary cause of hemorrhage. It is more probable that the constitutional effects of syphilis underly the degenerative changes in the blood and vessels, which lead to hemorrhage. The maternal evidences of infection are often obscure; the infection may be transmitted directly from the father and present no evidences in the mother, or the history may be misleading on account of the ignorance of the parents. In a case reported by L. E. Frankenstall (*American Journal of Obstetrics,* Vol. XLV., p. 270), it appeared reasonable to connect the hemorrhagic condition in the infant with the existence of tabes in the father, an antecedent condition which would have been entirely overlooked if the history had not been carefully investigated.

As to frequency, among 3,364 children born at the Philadelphia Lying-in Charity, between the years 1901 and 1904 there were forty-five instances of hemorrhage, the majority of which were fatal. Among this number of infants there were ten in whom the history of syphilis was either obtainable in the parents,

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or the clinical or postmortem evidence of syphilis was present in the children themselves.

Leakage may occur from the minute blood vessels in the skin, mucous surfaces, the serous membrane, the meninges and brain, and the parenchyma of the various organs and glands. Hemorrhage may appear also from the conjunctiva, the ears and umbilical cord. The skin hemorrhages may be in the form of oozing from the capillaries, very often seen occurring from the roll of skin which surrounds the cord. Bleeding may occur also from fissures at the mouth or anus. It may take place in the vesicles in pemphigus or from the base of the vesicles after rupture. Extensive extravasations between the skin usually affect dependent portions of the body, as the feet and ankles, also those surfaces which are exposed to pressure after birth, as the dorsal region, the buttocks and the extensor surfaces of the arms.

Extensive hemorrhage in the lower region of the abdominal wall and the scrotum or vulva is consequent to the ligation of the cord. A review of the fetal circulation will recall the fact that interference in the current of the umbilical arteries will induce a backward pressure in the iliac arteries, which affects their distributing branches, resulting in extravasation in the regions supplied by the latter. This brings us to the question whether there may not exist a general heightened arterial pressure in utero as a result of obstruction in the placental circulation due to obliterative changes in the vessels. Not only are the placental vessels affected by the syphilitic changes but those of the cord as well. It is even conceivable that torsion of the cord, frequently observed in instances of intrauterine syphilis, may have the effect of increasing the resistance in the fetal circulation. As a consequence of these various factors we may conceive of a condition of heightened pressure which shows its result at the time of birth when the demands upon the circulation are suddenly increased.

The extent of the hemorrhage varies. In the skin the epidermal layer may be invaded by extravasation in the form of petechiæ. A more extensive hemorrhage is apt to occur in the corium, elevating the epidermis. The areas involved are the buttocks or flexor and external surfaces of the thighs, the feet or the whole of the lower abdominal region. The discoloration is marked, presenting a dusky violet hue limited by a hyperemic zone. The contrast between the areas of discoloration and the icteroid skin surrounding them, observed in the majority of cases,

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is striking. The subintegumentary tissues, in pronounced cases, is involved; the hemorrhage permeates the fat, following the course of the connective tissue trabeculæ. The tissues may be generally thickened by the blood which is effused within the fat and which, in severe cases, invades even the muscles.

As to congenital hemorrhage, it may appear in the skin, usually in the form of petechiæ. Hemorrhage from the cord and mucous surfaces may also make their appearance soon after birth. Probably the most frequent early hemorrhage is in the form of hematemesis, which may begin with the effort to dislodge mucus from the pharynx or with the vomiting of meconium in instances of premature attempts at respiration. Later hemorrhages may appear from the fifth to the twelfth day. In fatal cases the termination is reached in from three to five days after the beginning of the bleeding. In cases which end in recovery the oozing may last for two or more weeks with a gradual lessening of the amount of hemorrhage; or, more frequently, as in hemorrhage from the cord for instance, may be arrested suddenly. In intracranial hemorrhage the symptoms of pressure begin early. Cases attended with intense icterus, with fever and rapid emaciation run a rapidly fatal course. Those with sudden onset and extensive extravasation or loss of blood by internal bleeding are unfavorable. Hemoperitoneum and subarachnoid hemorrhage are the accompaniment of severe diathesis. They point usually to early death. Bleeding from the cord is not necessarily fatal. In instances of continuous oozing, even to the point of exsanguination, a sudden cessation of the hemorrhage may occur and the child begin to improve.

Internal hemorrhages vary in their form according to their situation. In the lungs the bleeding is in the form of small apoplectic areas distributed generally (usually in the upper lobes) or peripherally in the form of infarcts. The subserous hemorrhages are usually limited to minute ecchymoses, although, as an exception to this, subcapsular hemorrhages of the liver may be extensive; it is not uncommon to find postmortem a subserous extravasation overlying both the right and left lobes of the liver divided by the suspensory ligament in the mid line and limited on either side by the costal borders. Hemorrhages in the spleen and suprarenal and thymus glands are usually in limited areas. In intraperitoneal hemorrhage the blood is usually unclotted. In subarachnoid hemorrhage the blood is held in a loosely formed clot which breaks down into fluid blood with the lightest handling.

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Hemorrhage affecting the mucous membrane may be in the form of free bleeding, which usually does not undergo clotting, or it may be in the form of submucous ecchymoses. The latter are seen particularly in the stomach between the rugæ, also in the upper intestinal tract. Bleeding into the connective tissue may be more or less extensive. A more or less frequent site for this form of bleeding is in the sheath of the umbilical vein.

The tendency to bleeding hinges primarily upon the comparative heightening of arterial pressure in the newborn. This is an inheritance from intrauterine life and is probably due to the limitation of the circulation by the absence of the pulmonary circuit. The comparative thickening of the adventitia in the vessel walls is corroborative of the existence of high arterial pressure. It is possible that this condition of the blood vessels may serve as a protection against the danger of a sudden reduction in the course of the circulation which results from the ligation of the cord and which may be only partially relieved by the establishment of the pulmonary circulation. It is necessary, however, to look for a more direct cause of hemorrhage than excessive pressure, otherwise the more or less common interference with the circulation, as from pressure from prolapse or winding of the cord during labor, and after labor from ligation of the cord, would be followed by hemorrhage. The pathological condition of the vessels in syphilis is confined, according to Schutz (Fischl, *Archiv. f. Kinderhk.*, Vol. VIII., 1886-7) to a thickening of the walls due to an hypertrophy of the muscular layer.

In spite of these facts the arterial changes in syphilis are not sufficiently distinctive to account for the tendency toward hemorrhage, as far as the arterial system is concerned. At best the consideration of the arterial changes is somewhat wide of the mark, as the extravasation occurs (except in instances of hemorrhage from the cord and in some instances of intracranial bleeding) in the form of capillary oozing. It therefore seems more likely that the relationship between the character of the blood and the containing capillaries is disturbed. The coagulability of the blood is certainly reduced. The persistence of bleeding from the cord in spite of ligation is evidence of this. The benefit from the internal administration of drugs which are supposed to increase the coagulability of the blood, as calcium chlorid, has been demonstrated in instances where ligation was entirely without effect.

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Recent bacteriological research has established the infectious cause of purpura in distinct instances. It is doubtful, though, that the presence of microorganisms postmortem should point conclusively to their existence as a direct or primal cause of hemorrhage in a large number of cases. In a very interesting series of cases of infectious origin described by Hamill and Nicholson (ARCHIVES OF PEDIATRICS, September, 1903), cultures made from the spleen, the lungs, the umbilical vessels and the heart's blood revealed the presence of microorganisms which in all likelihood were present in the environment of the children—in the air, possibly from the milk, upon the hands of the nurse or in the water with which the children were bathed. Conspicuous among these organisms was the colon bacillus. Relative to syphilis, however, these findings would make one incline to the view expressed by Capps (*American Journal of Obstetrics,* Vol. XLV., 270, discussion) that it is possible during the last few days of life for various microorganisms, especially the colon bacillus, to pass from the intestinal tract into the blood. The organism is thus to be found both in the blood and viscera after death. What applies to the colon bacillus is likely also the case with the staphylococcus aureus and the bacillus aerogenes lactis, the former being found in typical growth in 4 out of 6 cases reported by Hamill and Nicholson. Under these circumstances it is at least worthy of emphasis that the presence of microorganisms does not exclude the possibility of syphilitic infection. Further than this, syphilis is likely to play a more prominent rôle in instances of purpura ending in recovery than is infection due to the presence of microorganisms, as the latter is usually fatal.

Speaking generally, it would likewise be an error to overlook in many cases of supposedly infectious origin, syphilis, for, among other causes, notwithstanding the fact that sporadic instances of purpura due to infection may occur, it is beyond the probabilities that an infection so grave as this proves itself to be in certain instances, and in which the organisms present are found to originate in the infant's surroundings, should attack the children singly; yet in 45 cases of those noted in the records of the Lying-in Charity mentioned above none of them came in groups, the only parallel cases as to the time of occurrence being those in an instance of twins (Case No. 237).

In the matter of diagnosis hemorrhage appears usually in children who do not present the characteristic signs of syphilis.

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Where this rule does not hold good the congenital manifestations, such as pallor, rhagades, shrunken skin, are more often observed than the hereditary manifestations, such as the characteristic eruption and snuffles. The more distinct types of the latter form of syphilis seem to make their appearance in cases in which the infection is more or less recent, while in the hemorrhagic cases the bleeding seems to be the result of certain constitutional changes due to remote infection. The clinical manifestations which accompany hemorrhage are various, depending upon the development of the infant as to prematurity, upon the degree of blood dyscrasia, and probably upon the thoroughness of the specific infection. In my cases the following course of events and their relationship to the histories was noted:—

CASE I.—Death following umbilical hemorrhage, with wasting and signs of cerebral pressure. Autopsy revealed intracranial hemorrhage. The mother gave a history of infection by a former husband.

CASE II.—Death in a premature infant following umbilical hemorrhage. Clinical evidences of syphilis during life in the form of vesicular eruption of the plantar surfaces. Autopsy:—Subarachnoid hemorrhage, infarction of both kidneys. A history in the mother of previous miscarriage in the third month.

CASE III.—Death from asphyxiation. Skin hemorrhages in the form of more or less extensive extravasation. Syphilitic enlargement of the liver, the abdominal circumference reaching 31cm. History of syphilis in the mother. The autopsy revealed nothing of interest beyond the liver condition.

CASE IV.—Death immediately following birth in a premature infant. Bleeding from the cord. Petechial eruption. Marked syphilitic degeneration of the placenta.

CASE V.—Ecchymosis of the serous membrane and minute apoplectic areas in lungs revealed by autopsy. Tubercular syphiloderm in palmar surfaces. Laryngeal obstruction and marked scaling of the skin during life.

CASE VI.—Hemmorhage from the mouth and cord complicated by icterus. Typical manifestation of congenital syphilis in the form of emaciation and pallor. Autopsy:—Chronic hepatitis and suprarenal hemorrhage.

CASE VII.—Hemorrhage from the cord. Death following inanition in the course of suppurating gummata of the scrotum and great toe. No autopsy.

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CASE VIII.—Stillborn infant. Gumma on the external border of both feet. Scleral hemorrhage. Autopsy:—Subarachnoid hemorrhage, enlargement of the thymus.

CASE IX.—Stillborn infant. Subcutaneous extravasation. Evidence of leakage from the cord after ligation. Bullous eruption of the palmar and plantar surfaces.

CASE X.—Stillborn infant. Cutaneous hemorrhage. Autopsy:—Pericranial and subperiosteal hemorrhage, subcapsular hemorrhage of the liver. Syphilitic periosteitis and necrosis of the cartilage of the nose, in the mother.

Perhaps the most interesting clinical accompaniment of hemorrhage is icterus. It is present in some degree in almost every case. According to one theory icterus is a primary condition, which is followed by hemorrhage due to its toxic effect. Excessive jaundice usually increases the gravity of the outlook. There is no doubt that the presence of bile in the blood decreases its coagulability, showing that the bile has probably a disintegrating effect upon the blood. It seems more likely, however, considering the large number of children affected with jaundice without consequent hemorrhage, that it is secondary to the blood changes that produce hemorrhages. It may be, on the other hand, that, in weakly children, the invasion of the blood by bacteria present in the intestinal tract may give rise to jaundice, the result of their toxic action, so that in some cases the icterus may be a mere accompaniment of the hemorrhagic condition.

As to the conditions found postmortem, Behrend (*Deutsche Zeitschr. f. Prakt. Med.*, Bd. V., pp. 289-301) found in a case with a clear history of syphilis pulmonary lesions consisting of subpleural effusions with white hepatization and numerous gummatous nodes. The infant was born with a petechial eruption. In 2 of the cases described in the foregoing portion of this paper, apoplectic points were found in the lungs. The combination of hemorrhages with other pulmonary evidence of syphilis in the lungs is, in the writer's opinion, rare. The only instance among the cases of congenital syphilis in the writer's experience in which gummata were found in the lungs was one without accompanying hemorrhage. Behrend mentions, also, in the case alluded to, a fatty degeneration of the pappillæ of the heart muscle.

Fischl, on the other hand, in a case of hemorrhagic syphilis found that the musculature of the heart was not markedly altered. Fatty degeneration was not apparent; the nuclei were sharply

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outlined and lightly stainable. Beneath the pericardium small hemorrhages were to be seen, penetrating at individual points into the muscular surface.

Syphilitic children with evidence of internal hemorrhage require prompt treatment. The effort should be to sustain their flagging powers of resistance. Oil baths, alternating with mercurial inunctions, are indicated. The child, however, should be kept in the incubator and disturbed as little as possible, except for the necessary measures of treatment. In fact, rest is the first indication. For fever the ice cap is indicated. In intracranial hemorrhage the latter is directly useful as a local measure. In extensive skin hemorrhages rubbing for the purpose of carrying out inunctions should be omitted. In severe cases of melena peristalis should not be encouraged. With this in view the nourishment should be given frequently in small quantities and dry warmth should be applied to the body.

The following drugs have been used internally:—Suprarenal extract, half grain doses repeated. Adrenalin solution, 1-1000, one drop, in repeated doses. Gelatin water, in the proportion of two drams to one pint. Fluid extract of ergot, one drop doses. Talley, in an interesting case in which continuous bleeding resulted from a puncture (alluded to above), noted a favorable result from the use of calcium chlorid in half grain doses given every two hours.

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NOTE ON THE DURATION OF THE PRODROMAL PERIOD IN RÖTHELN.

BY D. J. M. MILLER, M.D.,
Philadelphia.

How long is the prodromal period of rötheln? In measles and scarlatina the stage preceding the eruption is reasonably fixed, but of rötheln this can scarcely be said. For, although it is true that, on consulting the abundant literature upon the subject, it will be found that the majority of writers consider the initial symptoms of this disease to be of extremely short duration or even absent entirely, yet there are others who ascribe to rötheln longer and more definite prodromes. Steiner denies their presence altogether (observed none in 21 cases). Oesterreich, Vogel, Klaatsch, Park, von Nymann, Ellis (slight or absent premonitory stage, one of the "chief points" in the diagnosis) and Bendix saw no definite premonitory symptoms. Others, and they comprise the great majority of observers, assert that, in most cases, such symptoms are either wanting entirely, the eruption being the first indication of illness, or are of slight import and short duration, lasting from a few hours to one, or at most, and very unusually, two days. Cases exhibiting long or severe prodromes, they declare, are anomalous. Among those who hold to this view may be mentioned:—Von Jürgensen, Thomas (never more than ½ a day), Griffith (150 cases), Rott (17 cases), Heubner (few hours to ½ a day), Dawson Williams (most cases rash first symptom; in others, from 12 hours to 2 days; seldom delayed beyond 2d day), Baginsky, Eustace Smith (average 24 hours), Hardaway, J. Lewis Smith, Squire, Atkinson, Holt, Plant, Unger and many others, too numerous to mention. Among those, on the other hand, who, while admitting that the invasion period of rötheln is often absent and its symptoms indistinct, have observed in their cases longer and more definite prodromes than the writers just referred to, are Roth (½ to 3 days), Claussen (3 days; some cases 6 to 7 days), Cheadle (3 days in 7 out of 8; in 1 case, 6 days), Emminghaus (3 days), Lindwurm (4 days), Thiefelder and Mettenheimer (1 to 3 days), Meigs and Pepper (2 to 4 up to

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7 days), Hemming (3 to 4 days), Edwards (3 days, average of 166 cases), Monti and Trousseau (1 to 4 days; generally well-marked).

A careful review of the testimony of observers upon this question would seem to show that in most cases the prodromal stage of rötheln is either absent, or does not exceed 24 hours, but that, occasionally, it may be prolonged to 2, 3, 4, or even 6 or 7 days. In reference to those cases in which no initial stage was observed, a probable explanation, of some of them, at least, is that, as the eruption is usually first noticed on rising in the morning, brief prodromes may have occurred during sleep.

The writer, while he is in accord with the great body of observers that the initial symptoms of rötheln are usually of short duration and more or less indefinite character, is inclined to believe, from the study of some recent cases, that the prodromes are prolonged and distinct much oftener than is generally supposed; and that the reason they are so seldom accurately determined is because physicians rarely have the opportunity to personally observe patients during this period, but must rely upon the statement of nurses and mothers. This belief is based upon 3 cases in which the unusual opportunity was afforded of closely observing the patients from the appearance of the first symptoms to the outbreak of the eruption; and upon the fact that, among many instances of rötheln observed by the writer, in the only cases (viz., the 3 here reported) in which it has been possible to definitely fix the prodromal period, the initial symptoms had a longer duration than is usually attributed to that affection, although this, of course, may have been purely coincidental.

That the number of cases is extremely small, if not altogether too small, from which to draw conclusions, is probably true; but it must be remembered that a few cases carefully and accurately studied may be as valuable as a hundred where the opportunities for observation have not been so favorable. For these reasons it has been thought worth while to place upon record the following cases:—The first patient was a girl of thirteen, the writer's daughter, who had been ailing for three days with what was thought to be a mild influenza, *i. e.*, with malaise, headache, pain in the legs and joints, and some loss of appetite; along with these symptoms were slight conjunctival injection and pharyngeal hyperemia, a temperature of from 99° to 100° F., and slight sore throat. On the morning of the third day the post-mastoid glands

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were found enlarged and tender. The next morning (the fourth day) the patient awoke covered with the characteristic eruption, if such a word may be used, of rötheln. With the appearance of the latter, the temperature was 100⅔° F.; but fell quickly to 99⅔° F., and in three days the patient had practically recovered from a mild but typical attack of german measles. In this case, then, the prodromes, which were distinct and well marked, lasted about 72 hours, *i. e.*, the eruption appeared at the beginning of the fourth day. The other 2 cases occurred in a family where the writer is in almost constant attendance, and where the anxious and careful mother seeks his advice on the slightest deviations of her children's health. The eldest of these children, a boy of eight, first showed symptoms of what appeared to be a trifling cold, on the morning of April 2d. That night he had slight fever, although the temperature was not taken. The next day he complained of nausea, and said that his legs "felt light" and tired. At noon the temperature was 99.5° F., and that evening 100° F. All that day and the next he played about and seemed fairly well, although there was some languor, slight injection of the conjunctiva, hyperemia of the throat (but no subjective symptoms of sore throat), and occasional sneezing. The temperature on the evening of the third day was 100° F., and the posterior cervical lymph nodes were enlarged. On the morning of April 5th (the fourth day), the rash was first noticed behind the ear and on the face. The subsequent course was characteristic of rötheln, the patient quickly regaining his usual health. The initial symptoms in this patient, though slight, were unmistakable, lasting about three days, the eruption appearing at the beginning of the fourth day.

Exactly fourteen and one-half days, *i. e.*, on the fifteenth day after the boy's first symptoms, and eleven and one-half days from the appearance of the eruption, about 9 P.M. on the evening of April 16th, his sister, aged five, having gone to bed apparently well, was noticed to have fever. The next morning her temperature was 99.5° F., that evening the same. With this there were coated tongue, some languor, decided redness of the conjunctivæ and pharnyx, but no marked conjunctivitis or photophobia. The cervical lymph nodes were not enlarged. These symptoms continued until the 22d of April, the child playing about as usual, although the temperature was constantly a little above normal, 99° to 99⅔° F. On the morning of the last date, *i.e.*, on the 22d of

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April (the sixth day), the patient awoke well covered with the eruption of rubella and a temperature of 102°F. The attack, however, was mild, and the child promptly recovered. The prodromes in this patient were also quite distinct, though less pronounced than in the others, and lasted five and one-half days, the eruption appearing on the sixth day. All these children had suffered from measles, and all had been exposed to rötheln, and, in addition, had other symptoms that placed the diagnosis beyond question.

Opposed to the above experience should be mentioned that of Forchheimer, who observed in one of his own children some indisposition for a short time only before the eruption appeared. Koplik, on the other hand, who saw no prodromes in the majority of his cases beyond a few days of preliminary fever, relates 1 case in which suffusion of the eye preceded the eruption by four days, and 2 others, in one of which enlargement of the cervical lymph nodes was present for six, and in the second for fourteen, days prior to the eruption, although he seems to regard such slight affections as symptoms of the incubation, rather than of the prodromal, period. An interesting feature, not referred to in describing the last of the writer's cases, was the distinct remission after the initial symptoms on April 16th; during the second, third, and fourth days of the prodromal period, the symptoms were so slight that they could have easily escaped notice. Toward the end of the fifth day, however, there was a slight increase of fever (100° F.), and if the patient had been first seen then, her symptoms might have been regarded as beginning on that day. Emminghaus is the only author who speaks of this tendency to remission and exacerbation during the stage of invasion, and its occurrence may explain why in some instances prodromal symptoms are not observed.

In conclusion, may it not be of the prodromal period, as it is of the eruption and other symptoms of rötheln, that it is an affection less fixed and regular in its features than any of the eruptive fevers; hence the various ways in which it is described by various observers; hence, also, the confusion as to its symptoms, and the doubt, which still exists in some minds, as to its independent nature.

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REFERENCES.

von Jürgensen. Nothnagel's Spec. Pathologie u. Therapie, Wien, 1896, Vol. IV., 2, p. 143.

Thomas. Cyclopedia of Practice of Med., v. Ziemssen, New York, 1875, Vol. II., p. 143.

J. P. Crozer Griffith. Medical Record, New York, July 2, 1887, pp. 11 and 37.

C. Mettenheimer. Journ. f. Kinderkrankheiten, 1869, Vol. LIII., p. 273.

Rott. Aertzl. Intelligenz-Blatt, 1879, Vol. XXVI., p. 101.

O. Heubner. Lehrbuch der Kinderheilkunde, Leipzig, 1903, p. 316.

Dawson Williams. Medical Diseases of Infancy and Childhood, London, 1898, p. 94.

A. Baginsky. Lehrbuch der Kinderkrankheiten, Braunschweig, 1894, p. 174.

Eustace Smith. Practical Treatise on Diseases in Children, London, 1884, p. 30.

N. Filatow. Vorlesung. über ac. Infect. Krankheit. in Kindesalter, Wien, 1897, p. 423.

H. Koplik. Archiv. f. Kinderheilkunde, Stuttgart, 1900, Vol. XXIX., p. 336.

Boulloche. Traité des Maladies de l'Enfance, Paris, 1897, Vol. I., p. 209.

W. A. Hardaway. System of Medicine, Pepper and Starr, Philadelphia, 1885, Vol. I., p. 585.

J. Lewis Smith. Diseases of Infancy and Childhood, Philadelphia, 1879, p. 192.

Wm. Squire. Quain's Dictionary of Medicine, New York, 1885, p. 1,382.

Steiner. Arch. f. Dermatologie u. Syphilis, 1869, Vol. I., p. 237.

Oesterreich. Inaugural Dissertation, Leipzig, 1868.

A. Vogel. Practical Treatise on the Diseases of Children, New York, 1885, p. 428.

Roswell Park. Chicago Medical Journal and Examiner, 1881, Vol. XLIII., p. 130.

E. Ellis. Practical Manual of the Diseases of Children, New York, 1879, 3d edition, p. 45.

A. Trousseau. Lectures on Clinical Medicine, American Edition, Philadelphia, 1882, Vol. I., p. 186.

Atkinson. American Journal of the Medical Sciences, January, 1887, p. 22.

Digitized by Google

L. E. Holt. Diseases of Infancy and Childhood, New York, 1897, p. 926.

W. T. Plant. American Text Book of the Diseases of Children, Starr, Philadelphia, 1894, p. 176.

L. Unger. Lehrbuch der Kinderkrankheiten, Leipzig u. Wien, 1901, p. 411.

Roth. Deutsches Archiv. f. klin. Medicin, Leipzig, 1874, Vol. XIV., p. 537.

Claussen. Chicago Medical Review, 1881, Vols. III. and IV., p. 212.

W. B. Cheadle. Transactions Internat. Med. Congress, London, 1881, Vol. IV., p. 4.

H. Emminghaus. Handbuch der Kinderkrankheiten, C. Gerhardt, 1877, Vol. II., p. 334; also, Jahrbuch f. Kinderheilk., N. F., 1871, p. 56.

Lindwurm. Bayer, Intelligenz-Blatt, 1867, n. 40, p. 585.

Thierfelder. Greifswald. Medicinische Beiträge, 1864, Vol. II., p. 14.

Meigs and Pepper. Diseases of Children, Philadelphia, 1877, p. 896.

W. D. Hemming. Edinburgh Med. Journal, 1880, Vol. XXVI., p. 52.

W. A. Edwards. American Journal American Sciences, October, 1884, p. 452.

A. Monti. Kinderheilk. in Einzeldarstellungen, Berlin u. Wien, 1901, Vol. II., p. 543.

Forchheimer (Cited by Koplik). Archiv. f. Kinderheilk., 1900, Vol. XXIX., p. 336.

von Nymann. Jahrbuch f. Pædiatrik, N. F., Vol. IV., Bd. ii., p. 123.

B. Bendix. Lehrbuch der Kinderkrankheiten, Berlin u. Wien, 1903, p. 571.

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UMBILICAL CORD HERNIA.

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Hernia at the umbilicus in early infancy may be of two forms.

(1) First the acquired, in which a portion of the intestine projects through the rectus muscle, and pushes before it a layer of peritoneum and the tissues of the skin. This variety is so well-known, and of so frequent occurrence that no further description is required.

(2) The second form is that known as congenital hernia, or hernia into the umbilical cord (hernia funiculi umbilicalis). This is sufficiently unusual to justify me in exhibiting to you the specimen before you. It occurred in the practice of Dr. Weiszgerber, and was kindly given me by him. It is a fetus, which was born at full term, and is, as you see, a monster. The vault of the cranium is entirely absent, and the brain is exposed. The matter to which I wish to call especial attention is the condition of the navel itself. When this specimen was fresh there was situated here a translucent tumor, which was about the size of a quite small apple, and contained some loops of intestine. At the base of the tumor, and surrounding it, was a narrow circular area composed of tissue resembling in appearance that of the mass itself; *i.e.*, a translucent membrane, in which there was evidently no trace of muscle or true skin.

Without entering at all upon the details of the literature of the subject, hernia into the umbilical cord consists of the projection of a portion of the intestine, or even more of the abdominal organs through an opening at the navel. This opening is the result of arrest of development of the abdominal walls, but the original cause of this failure of development is uncertain, and various theories have been entertained.* The hernia forms a tumor varying in size from that of a nut up to that even of a newborn infant's head, or larger. It is oval, round or barrel-shaped, with the cord starting

* A statement of the various theories, together with a useful bibliography, may be found in a careful article by Dr. Wollstein in the ARCHIVES OF PEDIATRICS for June, 1904.

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from the distal portion. The hernial sac appears to be composed of the distended umbilical cord. Its walls consist of peritoneum and of the amnion of the cord alone, and are grayish white and translucent. The contents are usually coils of intestine, but sometimes all, or a part, of the liver, the stomach, the spleen, or other organs may be found in it. The color of these is distinguishable through the walls of the sac. The size and tension of the mass increase with crying or coughing. The hernia can sometimes be reduced, and sometimes not. When it is of small size, recovery may take place spontaneously with the removal of the cord. A reactive inflammation then sets in around the ring-shaped border of the hernia. The color of the hernial sac changes, the umbilical cord shrinks, and, finally, it and the amnion separate. Granulations then spread gradually over the surface remaining; generally with free suppuration. As the wound thus left heals and shrinks, the hernia disappears within the abdominal cavity. A cicatrix remains, but no real navel.

Many dangers, however, attend this process, and a fatal issue generally results. The peritoneum is very liable to invasion, the hernia becomes gangrenous, or general sepsis develops. The prognosis is, on the whole, grave. By far the larger proportion of cases died until the radical operation was introduced and perfected. Even thus the mortality is large.

In case of very large hernias with the presence in the sac of a considerable portion of the abdominal contents the subject is scarcely able to live. Sometimes the sac breaks during birth, leaving the child partly eviscerated. Very often the infant exhibits other malformations, some of which are incompatible with life, as in the case exhibited this evening. Not infrequently the infants are premature or stillborn.

The diagnosis is easy, except in the case of small cylindrical hernias into the cord. Here it is easy to overlook the condition, and to apply a ligature, which, of course, ligates the intestine as well. Every child, born with that portion of the cord which is next to the body decidedly swollen, should be examined very carefully before a ligature is applied.

The treatment consists, first of all, in the greatest care in handling a congenital hernia, and in the use of every possible antiseptic precaution. If the hernia is small, and if reduction can be made quite easily, this may be done, and an antiseptic compress applied and held in place with adhesive plaster. The child must not be

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lifted into an upright position until the wound has completely cicatrized, and this may not be for weeks.

If the hernia is not reducible, it may be covered with an antiseptic protective dressing, in the hope that granulations may form, and the process go on as just described. A much more successful plan of treatment, however, applicable, also, to the small reducible hernias, is the doing of a radical operation as soon after birth as possible, and without any previous effort at reduction being made. The operation may be done either without the opening of the peritoneum or with it. After the operation the temperature of the child should be maintained, preferably in an incubator. Many infants have been saved by the operative procedure.

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CONGENITAL OBLITERATION OF THE BILE DUCTS.

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This is one of the uncommon causes of icterus in the newborn, yet a number of cases have been reported. There have been several collective investigations made, among them those by Giese (*Jahrb. f. Kinderh.*, 1896, Vol. XLII., p. 252) and Gessner (*Ueber Kongen. Verschl. der grossen Gallengänge,* Dissertation, Halle, 1886), but the most noteworthy contribution to the literature of the subject is that by John Thomson ("Congenital Obliteration of the Bile Ducts," 1892). This writer tabulated 49 cases verified by autopsy, and added one of his own. In a later publication (Allbutt's System of Medicine, 1897, Vol. V., p. 253) he estimates that not more than 60 or 70 cases have been published. Among other serviceable articles of comparatively recent date are those by Ralleston and Hayne (*British Medical Journal,* 1901, Vol. I., p. 758), Skormin (*Jahrb. f. Kinderh.*, 1902, Vol. LVI., p. 196), F. Parke Weber (*Edinb. Med. Journ.*, 1903, Vol. LVI., p. 111), Arkwright (*Edinb. Med. Journ.*, 1902, Vol. LVI., p. 156), and Mohr (*Ein Fall v. Kongen. Icterus*, etc., Dissertation, Berlin, 1898). A more extensive bibliography will be found in some of them. My own case, of which the notes are necessarily meagre, owing to the shortness of the time of observation, is as follows:

Fred Corbin. Age ten days. The family history is entirely negative, no similar disease having occurred in it. The child was born at term, apparently well. Meconium was passed per rectum until the age of three days. Some infection of the cord now appeared to be present; the stump fell about the eighth day. On the third day after birth the mother noticed jaundice. With this was associated frequent vomiting, constipation, and loss of appetite. These symptoms grew more severe, the juandice becoming a deep yellow, and the baby was brought to the Children's Ward of the Hospital of the University of Pennsylvania, May 23, 1903, on the tenth day after birth, or the seventh from the time the symptoms commenced.

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Examination showed a small, emaciated infant. The skin and conjunctivæ were extremely icteric; the pulse was feeble and rapid; the respirations were accelerated. Vomiting was very frequent, the child being unable to retain any food swallowed. When given by the stomach tube food was rejected. Constipation was absolute. Bile was present in the urine.

Death took place twelve hours after admission, there having been, in the meantime, no movement of the bowels. A short time before death general edema appeared and became very marked.

While in the hospital the temperature varied from 96.8° to 99.2° F., the pulse from 140 to about 155 and the respiration was about 42.

Autopsy.—The autopsy showed a complete obliterating stenosis of the common bile duct situated about one-quarter of an inch above its opening into the intestine. The duct at this point was hard and cordlike, and could be easily rolled between the fingers. No bile could be forced through it from the gall bladder, although the duct above the stenosis could be easily distended by pressure on this viscus. The gall bladder was of normal size and filled with bile. The surface of the liver and the intestines in the vicinity were stained with bile, from contact with the bladder. The liver on section was of normal color. There was no bile in the duodenum. The pancreatic duct was patulous.

A microscopical study of the liver was made by Dr. C. Y. White, who reported as follows:

"The liver shows a moderately severe grade of fatty degeneration of many of the cells, and cloudiness of the remaining ones. In some sections there is a slight increase in the amount of fibrous tissue surrounding the bile ducts, but not to any noteworthy extent. In other sections there is certainly none. The intrahepatic bile ducts are small, and the majority contain granules of bile pigment. The common duct above the obstruction is patulous, and the walls show a good deal of fibrous tissue."

Pathological Anatomy.—The lesions characteristic of this disease consist in obstruction somewhere or other in the course of the biliary passages. There may be no discoverable trace of the bile ducts, or of a portion of them, or they may be represented by a fibrous cord only. The gall bladder may be absent or may be much dilated. In some cases the ducts are apparently normal in size and position, but there is a total obliteration of their lumen

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at some one point; or, the ducts may be dilated above the point of obstruction. The lesions may affect one or all of the main bile ducts. The liver is generally enlarged, firm, and either of a natural or a dark green color, and exhibits lesions of hypertrophic cirrhosis. In the case now reported there was, however, no noteworthy degree of cirrhosis present. The blood vessels are seldom affected. The spleen is enlarged. If the case has run a chronic course, the liver may exhibit atrophy.

Pathogenesis.—The cause, often obscure, appears to be of different nature in different cases. In many cases it is possible that there has been some influence acting, which prevented the proper development of the ducts during fetal life. It is a striking fact that the disease is liable to occur in several members of the same family. This supports the theory of some congenital failure in development. In other cases the existence of an obliterative inflammation seems probable, although the cause and place of origin of this is uncertain. According to some views this inflammation starts as a cholangitis within the liver itself, and descends to the main bile ducts. The development of the cirrhosis goes *pari passu* with the cholangitis. Another view places the cirrhosis as a lesion secondary to the complete obliteration of the ducts. The case I report would support the latter view. There had not been time for a secondary cirrhosis to develop to any extent. In some cases there may have been a fetal inflammation of the mucous membrane of the ducts. The narrowing thus produced may not effect a complete obstruction at first, but a catarrhal swelling results, which finally stops entirely the passage of bile. The inflammation may then spread to the walls of the duct, and complete atresia follow. This may take place either before birth, or after it. The irritation of a gall stone in the duct may rarely have been the starting point of the inflammation. It is also conceivable that the origin of the inflammation may be syphilitic, and that there has been a gummatous deposit in the bile passage. Yet if this is so we ought, as Thomson points out, to find associated signs of syphilis elsewhere. In fact, there seems little evidence that congenital syphilis plays a role of importance in the etiology.

Symptoms.—The chief symptom is icterus. This is present at birth, or develops within the first week, less often in the second week or later. In a case reported by Köstlin (*Canstatt. Jahrsber.*, Vol. III., p. 293) it did not appear until the age of six months. The color is intense and persistent, and often of a greenish hue.

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The second characteristic symptom is the occurrence of acholic stools. This may be present from the beginning, or there may be at first evacuation of normally colored meconium, if the blocking of the ducts has occurred late in fetal life. In such cases the blocking cannot be the result of an arrest of development. It is even possible that ordinary yellow movements may be present for a time, and these give way later to acholic stools; five weeks in a case reported by Skormin (*l. c.*) This is, however, of rare occurrence, and can only be explained on the ground that the congenital narrowing did not become complete obliteration until a considerable time after birth. The urine is intensely bile stained. Fever is not a symptom. Vomiting is apt to occur. Hemorrhages from the umbilical cord, and into the different parts of the body constitute a very characteristic symptom. The children do not, as a rule, show any evidence of malnutrition at birth, but this eventually develops if the case is prolonged. Death occurs in convulsions or sopor or from exhaustion. The course of the disease is seldom acute. The fatal ending comes generally only after several weeks, or even months, although it may occur in the first week of the disease.

Diagnosis.—This rests chiefly upon the persistent jaundice and the presence of acholic stools, yet the recognition of the disease is not so easy as it would at first sight appear. Cases of simple icterus neonatorum may sometimes be extremely persistent and intense, and the diagnosis from obliteration of the bile ducts, may be in doubt for some time. I recall one instance particularly in which jaundice lasted several weeks, and in which for some time I was in doubt. The stools were whitish and glistening, and clearly contained an excess of fat, and apparently a deficient amount of bile. Bile, however, was proved to be present. There was also persistent fever, although sepsis could be excluded with tolerable certainty. Recovery took place slowly.

A tendency to hemorrhage in various parts of the body is a characteristic of other forms of intense persistent jaundice, as well as of the one under consideration. It is quite frequently associated with jaundice as a symptom of sepsis in the newborn. In these cases, however, the existence of fever and of stools showing the presence of bile, together with the early presence of symptoms of severe icterus, aid in excluding congenital obliteration of the bile ducts as the pathological condition present.

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In this connection, too, attention must be called to the fact that a family predisposition may exist in other forms of icterus. This has been emphasized in the very instructive paper by F. Parkes Weber (*l. c.*). He points out the fact that Hanot's Disease (biliary cirrhosis with chronic jaundice) may occur in different members of one family. He also describes a class of cases which he designates "Simple Persistent and Congenital Persistent Jaundice," which he believes probably due to an obliterative inflammation of bile ducts analogous to, but of more limited extent than, that present in the cases of Thomson's type. Thomson, too, refers to fatal cases with the symptoms of obliteration, but with patulous ducts found at autopsy. He regards them as examples of different stages of the same disease.

In "Congenital Obliteration of the Bile Ducts," as described by Thomson, there is nearly always present also a congenital biliary cirrhosis, as already pointed out. Rolleston and Hayne (*l. c.*) believe that the cholangitis and the cirrhosis developed at the same time, and that the former, starting within the liver, descends to the larger hepatic ducts, producing, finally, occlusion of these. If this is true, it places the condition, in many respects, very close to Hanot's Disease. The case now reported does not, however, support this theory.

The difficulty, then, in distinguishing congenital obliteration of the bile ducts from other forms of severe icterus, fatal or otherwise, becomes evident. The cases described by Arkwright (*l. c.*) illustrate this well. In these, fourteen of the fifteen children of one mother, suffered from icterus soon after birth and ten died. In two of them, however, the icterus disappeared before death, and in none of them is it certain that the stools were typically acholic. Some of the cases suggest strongly in most respects obliteration, and the probability is great that the diseased condition was identical in all of them. Yet the diagnosis of obliteration cannot be made, if we consider the disease to be uniformly fatal. The cases of Pearson ("Underwood's Diseases of Children," Tenth Edition, 1846, p. 168), in which of eleven children of one mother, ten died of jaundice soon after birth, and one of jaundice at six years, are all strongly suggestive of obliteration. Yet in the absence of autopsy the diagnosis cannot be certain, and the question also arises, as to the nature of the lesion in this six-year-old girl.

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In this connection I wish to detail the salient points of the history of a case of icterus seen with Dr. J. E. Talley and already reported before this Society. (See ARCHIVES OF PEDIATRICS, September, 1904.) The infant was the fifth child of its parents. The first three are alive and well. The second suffered from decided jaundice soon after birth, which lasted for weeks. The fourth child had intense icterus, and died about the third day of life, no other cause for death being discoverable. The patient under Dr. Talley's care developed jaundice shortly after birth. This became intense. The urine was bile-stained; the stools were dark-colored. There was no fever. On the tenth day a widespread eruption of purpuric spots and subcutaneous hemorrhages developed. There was a trace of blood from the umbilicus at about three weeks of age. A puncture of the toe for a blood-examination, made when the child was a month old, was followed by hemorrhagic oozing, which lasted for a week and was controlled with the greatest difficulty. The purpuric spots were still appearing. The jaundice had been lessening. By six weeks from birth the jaundice was much better, and few new cutaneous hemorrhages were developing. The stools soon after this became a yellow color.

This case, from the family history and the severity of the very characteristic symptoms, points strongly to the presence of obstruction of the bile ducts, which had not yet reached the degree of absolute obliteration, since the stools appeared to contain bile. The fatal case in the family could properly be considered an instance of the disease, so far at least as the diagnosis can be made from the symptoms. Concerning the one which recovered previously, we can hardly form a conclusion. The case seen by us would have received this diagnosis had it ended fatally. The result proves that if obliteration were present this could not have been of a permanent nature.

Although, then, the inflammatory process which in fetal life or in the first days of the existence of the newborn, though not causing complete obstruction at once, usually tends to grow worse, may it not be equally possible that in rare cases the progress may instead be arrested? It would seem at least possible that a congenital narrowing, due to defect of development or to a cholangitis, did, in fact, exist in Dr. Talley's non-fatal case; but that the actual severe obstruction, with consequent symptoms, was of a nature susceptible of recovery, as, for instance, a

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catarrhal inflammation, which completely closed for a time an already narrowed duct. This would be a condition entirely analogous to that probably present in some of the cases of congenital stenosis of the pylorus.

Prognosis and Treatment.—The prognosis of cases of obliteration, if this is complete and organic, is absolutely bad, and treatment can be only palliative unless the thought of operative procedure be entertained. An operation, however, of this gravity on subjects at such an early age appears to offer little hope.

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REPORT OF A CASE OF PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

BY HOWARD CHILDS CARPENTER, M.D.,

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The patient, Joseph N., aged fifteen years, was admitted on January 7, 1905, to the Philadelphia Orthopedic Hospital, in the service of Dr. William J. Taylor, to whom I am indebted for the privilege of reporting the case. The history is as follows:

Family History.—Father died at the age of twenty-five years from pneumonia. Mother is living and well. One other child, a sister, aged sixteen years, is living and well. No history of pseudohypertrophic muscular paralysis in family.

Personal History.—The child was born asphyxiated, after a difficult labor. As an infant he was always delicate. When four years old, glands, probably tubercular, were removed from the right side of his neck. Following an attack of measles, when five years old, the patient was ill for two years. His mother says he had consumption. (?) But previous to the measles the mother noticed that he was extremely weak in the lower extremities, and when walking, if given but the slightest jolt, would fall over. She also noticed that it was very difficult for him to rise from the ground; in fact, it was impossible for him to do so, unless he turned over on his abdomen, and pushed himself upward by his arms. Since that time, this weakness has increased steadily, until now he is scarcely able to walk, even on the level. Three years ago the patient was in the Children's Hospital, for about eight weeks. The diagnosis was made at that time of pseudohypertrophic muscular paralysis. While in the hospital he had a severe attack of pleurisy. He began to attend school when seven years old, but made very little progress, for five years he remained in the first grade. Although feeble, his mental processes are normal. He is able to read, and writes very well.

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Physical Examination.—The child's appearance is most striking. His station, gait, and muscular development leave no doubt as to the diagnosis. His general appearance is that of a child about nine years old. His head is somewhat flattened antero-posteriorly. The pupils are equal, and react to light and distance. His teeth are in poor condition, and there is a scar on the right side of neck, below the angle of the jaw.

Over the entire body the skin has a mottled appearance, the hands are somewhat cyanotic, with slight clubbing of the fingers, and on the left arm there are scars of three successful vaccinations.

He is able to walk alone only with difficulty; he has the characteristic waddling gait, with feet far apart and toes turned in. He is able to stand alone only when his feet are widely separated. His station is typical, with a marked lordosis, but there is no lateral deviation of the spine. The boy has a rachitic chest with a tendency to a pigeon-breast, and beading of the ribs.

FIG. I. CASE OF PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS. PHOTOGRAPH SHOWING ATROPHY OF SCAPULAR MUSCLES, LORDOSIS AND ENLARGEMENT OF CALF MUSCLES.

But the most striking feature is his muscular development; the enormous hypertrophy of the calf muscles, the soleus and gastrocnemius; the tibialis anticus is also hypertrophied. These muscles on palpation are firm. The circumference of the mid-calf on right side is 29 cm., while on the left side it is 27½ cm. When the patient was in the Children's Hospital, three years ago,

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the mid-calf measured 28½ cm. There was no mention made whether this was the right or the left calf.

At present the circumference of the mid-thigh on the right side is 33½ cm., and on the left 33 cm., while three years ago, the measurement was 32 cm. There is slight talipes equinus, the foot cannot be brought up more than a right angle.

The length of the right lower extremity, from the anterior superior spine of the ilium to the internal malleolus, is 67 cm., while the left lower extremity is one centimetre shorter, measuring 66 cm.

FIG. II. PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS. PHOTOGRAPH SHOWING ENLARGEMENT OF POSTERIOR AND ANTERIOR LEG MUSCLES.

Both knees have complete flexion and extension.

The gluteal muscles are well developed, but not over developed; whereas the quadratus lumborum is over developed on both sides equally. The entire shoulder joint may be raised to the top of the ear, showing the marked wasting of the trapezius, latissimus dorsi, pectoral muscles, deltoid and scapular muscles, with the exception of the infraspinatus, which is hypertrophied. The muscles of the neck are not affected. The biceps and triceps are wasted, but wasting is not so marked in the forearms, where flexion, extension, pronation and supination are good. The hand muscles are fairly well preserved. Some slight wasting of the interosseous muscles and the thenar eminence.

The dynamometer shows very feeble power in the hands; slightly more strength in right hand than in the left.

The apex beat of the heart is one inch below the nipple, and just within the nipple line. The heart is not enlarged. A systolic

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hemic murmur is audible. The liver and spleen are not enlarged.

The patient's average morning temperature is 99°F., and the afternoon temperature is 99.5F. Average respiration is 24, and average pulse is 98. There is no albumin or sugar in the urine. The knee jerks are not diminished. Sensation is normal.

There are no reactions of degeneration in the arms or legs, and faradic response is good.

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A FATAL CASE OF ACUTE RHEUMATIC ENDOCARDITIS WITH RHEUMATIC SYMPTOMS IN JOINTS AND MUSCLES BARELY NOTICEABLE.

BY EDWIN E. GRAHAM, M.D.,

Professor of the Diseases of Children in the Jefferson Medical College, Philadelphia.

I was called on January 11th by Dr. C., to see the child of Dr. S., aged four and a half years, and obtained the following history:—The boy had, during his first year, suffered from a mild attack of ileocolitis, otherwise he had always been strong and healthy; the family history was good, no rheumatic or tuberculous tendencies. December 13th the boy had been taken on a sleigh ride a few miles in the country by his father. It was a clear, dry, crisp morning, he was well bundled up, and was in apparently the best of health. Upon their return home, the boy complained of feeling rather stiff, when lifted out of the sleigh, and during the subsequent week he complained of slight stiffness, but not enough to keep him off his feet, in fact, he played with his toys and acted very much as was his usual custom. Twenty-four hours before my visit, a slight cough developed; his urine became scanty, containing considerable albumin and one or two hyaline casts. When I saw him at 8 P.M., on January 11th, his temperature was 101° F., respiration 40, pulse 150. He complained of little or no pain, his limbs could be moved with little or no discomfort, and his mother informed me that about the only time he had complained was during the day, in stooping down to lift some toys from the floor, when he spoke of some slight stiffness in his legs.

The lungs were clear. In the mitral area, the first sound of the heart was entirely replaced by a soft, blowing, mitral murmur. The second sound of the heart in the mitral area was clear and distinct. This murmur was transmitted well to the axillary line and to the angle of the scapula. It became fainter at midsternum, and still fainter at the aortic cartilage. The amount of urine passed had increased since the examination made twenty-four hours previously; the albumin had almost disappeared and no casts could be found. The case was evidently, one of acute rheumatic endocarditis where the inflammation, with all its violence, had at-

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tacked the endocardium in the region of the mitral valve, rather than the joints of the body. The boy was unusually sturdy and well developed, and previous to the present illness he had never suffered from dyspnea on exertion; in fact, he had been able to romp and run with the best of his associates; he had never had cyanosis; had never had any evidences of rheumatism, and previous to the present illness he had never suffered from any infectious disease, which could affect the endocardium. The conclusion seemed to me to be that the endocarditis was a primary endocarditis, that it was a simple endocarditis, not a malignant endocarditis, and that the inflammation in the mitral leaflets was of an unusual severe character. The rapid respiration, cough, scanty urine and albuminuria were due, undoubtedly, to the congestion resulting from the heart lesion.

The boy's clothing was removed at once, and he was placed in bed in the charge of a competent trained nurse, and was kept absolutely at rest. The bowels were moved by fractional doses of calomel followed by a saline; he was given liquid nourishment in the shape of milk every three hours and an abundance of water. Five grains of salicylate of soda were administered every fourth hour, and one drop of veratrum viride every three hours. The next day, Wednesday, at noon, the temperature had fallen to 99½° F.; the heart action was slower, 140; the cough less; the dyspnea was less, and the urine was being passed in larger amounts, and the boy seemed distinctly more comfortable.

Friday morning, at 12 A.M., he awoke out of a dream very much frightened and excited; his shortness of breath became very much worse, dyspnea became marked, and he died at 1 A.M., one hour later.

The case seems to me to be of unusual interest for a number of reasons. The rheumatic symptoms were of such a passing character. The boy was able to be on his feet until about thirty hours before his death. The endocarditis was almost the only manifestation of rheumatism. It was simple endocarditis, and not malignant. The early termination in death in exactly one month.

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INFECTIVE ENDOCARDITIS IN A BOY TEN YEARS OLD: RECOVERY.

BY CHARLES F. JUDSON, M.D.,
Philadelphia.

Infective, malignant (so-called ulcerative) endocarditis is a rare disease in childhood. Various types of this malady have been described as the septic, typhoid, cerebral and the cardiac, according to the predominance respectively of septic, typhoid, cardiac and cerebral symptoms. The cardiac type of infective endocarditis, which is the most frequent, may run a subacute or chronic course for several months or even a year. Although recovery is extremely rare, several undoubted cases have been put on record. For this reason it seems worth while to report my observation of the disease in a boy aged ten years who recovered after an illness lasting over three months.

The microorganisms usually found associated with the lesions of acute, infective endocarditis are the staphylococcus, streptococcus and the diplococcus pneumoniæ. More rarely we find the typhoid bacillus, gonococcus, diphtheria and tubercle bacillus. Infective endocarditis may be primary or it may complicate septic processes, or it may occur secondarily in the course of the acute infectious fevers, pneumonia and acute rheumatic endocarditis. Rheumatic fever much more commonly gives rise to the ordinary type of endocarditis with verrucose vegetations, but destructive processes of malignant nature undoubtedly occur in this disease.

Ogle has reported 3 cases of this type. Traumatism may rupture a heart valve and lead to ulcerative inflammation. Sclerotic changes in the valves also predispose to this disease. In Coupland's series of 69 cases, 61 showed signs of previous disease; in Kanthack and Tickell's series of 84 cases 68 had old cardiac lesions. The disease is rare in childhood. Of 209 cases collected by Osler only 4 occurred in children. In Osler's series the aortic and mitral valves were affected together in 41 cases, the aortic valves alone in 53, the mitral alone in 77, the tricuspid in 19, the pulmonary valves in 15 and the heart wall in 33. The pulmonary valves were the seat of disease in 4 instances, the tricuspid in 5 instances. Beside the heart lesions and those of the primary dis-

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ease, the most characteristic pathological findings are metastatic abscesses and infarcts due to embolism.

The subject of the present report is Joseph B., a school-boy, ten years old. His mother died of tuberculosis, and two of her children died in infancy, one of marasmus and one prematurely born. Joseph had been well until the present illness, excepting an attack of summer diarrhea, measles and occasional sore throat. Two weeks before his admission to the Episcopal Hospital he suffered from headache and pain in both knees, followed by nausea and vomiting which lasted two days. Chilly sensations were followed by fever of sudden onset. At the same time a masculo-papular rash appeared about the hands and spread over the trunk and limbs. When admitted to the hospital this eruption, which was very profuse, consisted of dull red scattered papules, with here and there a bright red central area, fading partly on pressure and like the lesions of urticaria pigmentosa, leaving behind a discolored brownish spot. The eruption came in crops, caused no itching, and was a prominent feature of the first two weeks of his illness. On admission there was no visible swelling or redness of the joints, but pain was felt on manipulating the knees. The arthritis spread to the left ankle, left elbow and right ankle within the next three days. Two days later the right wrist, metacarpo-phalangeal joint of right index finger and the first joint of the little finger were involved. The condition of the heart when first examined was fairly good, the action was rapid and regular, and the impulse fairly strong. The apex beat was in the fourth interspace within the nipple line and not diffuse, there was no thrill, the first sound was reduplicated at the apex, and a soft systolic murmur was heard over the whole precordia loudest at the pulmonary cartilage and not transmitted. Cardiac dulness was normal. The lungs were clear. There was no demonstrable enlargement of the liver or spleen, and there were no abdominal symptoms except slight anorexia. The mind was perfectly clear. Cephalalgia was moderate. The urine was passed freely, its specific gravity was 1.010; it contained a trace of albumin, but no casts. The clinical picture and the irregular continued fever suggested at first acute articular rheumatism with involvement of the heart, and salicylates were given freely but with little benefit. The temperature toward the end of the third week of the disease began to show the wide variations of infective ulcerative endocarditis. Slight sweating was noticed, the rash became more dusky in color,

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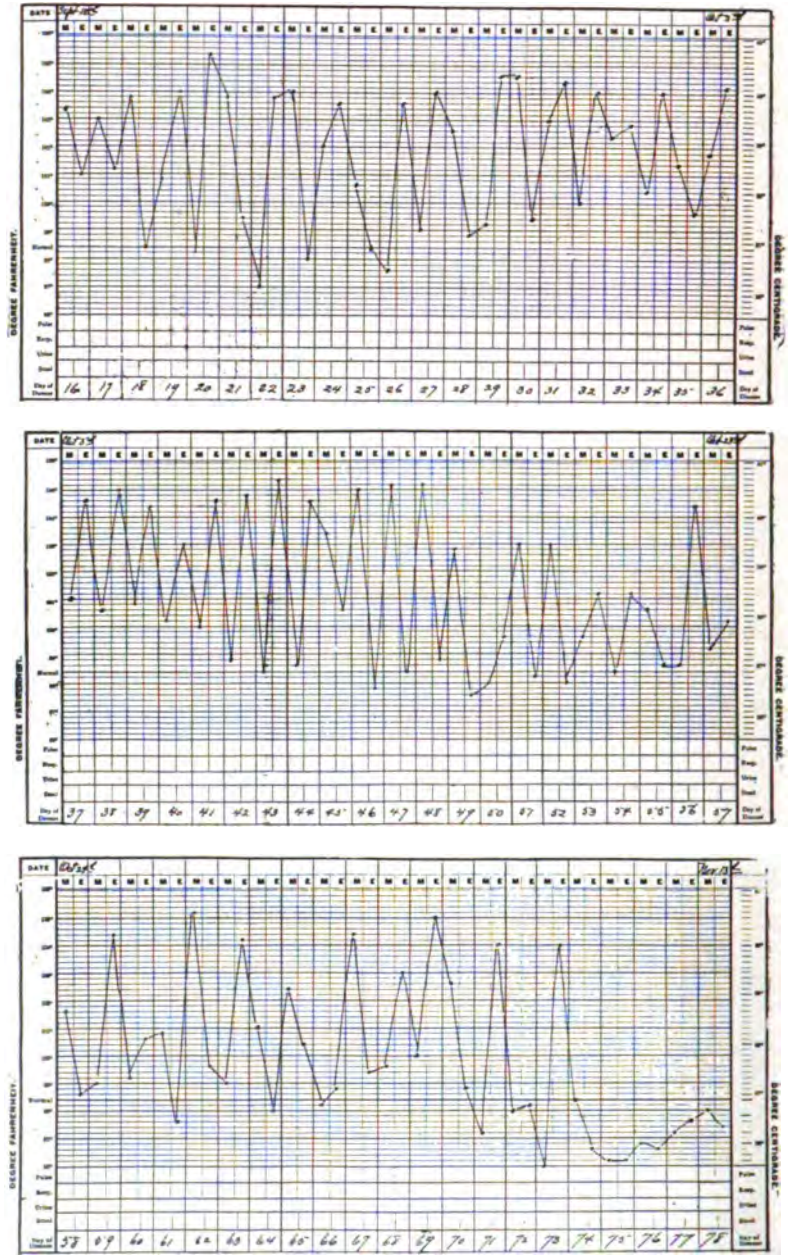

TEMPERATURE CHART IN CASE OF INFECTIVE ENDOCARDITIS. RECOVERY.

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and signs of cardiac dilatation developed. The systolic murmur became louder and rougher. The spleen was demonstrably enlarged. The urine reacted for albumin, and contained many hyaline and a few granular casts. A culture taken from the blood three days after admission and repeated three weeks later gave a diplococcus in pure culture. The leukocytosis was marked, ranging from 20,000 to 34,800. There was from the first marked anemia which became progressively worse. Examination of the eye grounds by Dr. Van Pelt showed no gross changes in the fundus (no hemorrhages or areas of degeneration), but there was slight tortuosity of the retinal vessels, and the margin of the disk was hazy. Intravenous injections of 3-5 cc. collargol in 4 per cent. solution were administered during the fifth week of the disease, besides inunctions of unguentum Crede. In spite of vigorous stimulant treatment the heart became gradually weaker, edema developed about the feet and soon became general, and the long bones were tender on percussion. The red cell count fell to 1,745,000 and the hemoglobin to 21 per cent. on October 8th, in the sixth week of the disease. The urine became scanty and albuminous, with hyaline and granular casts. The patient slept most of the time, and was irritable when aroused. The heart sounds were at times almost inaudible. The seventh, eighth and ninth weeks were marked by extreme prostration, general edema, ascites and scanty secretion of urine, much below the intake of fluids. The fever had diminished in intensity by the eighth week, and intermissions of several hours duration occurred. On the last day of October, the sixty-fifth day of illness, diuresis suddenly set in, with the excretion of ninety-five ounces of urine and continued for one week, causing the disappearance of the edema of the legs and scrotum. Theocin had been given in small doses (2 grains) for five or six days previous to the day when the secretion of urine became copious. Improvement was continuous during the next two weeks. The temperature reached normal on the seventy-sixth day and took but one sudden rise after this, on the seventy-ninth day. The signs of cardiac dilatation had largely disappeared three days later, but a faint systolic murmur was still audible over the base of the heart, with reduplication of the first sound.

The chief points of interest in this case concern the diagnosis and treatment. Although the multiple arthritis suggested rheumatic fever, the typical temperature curve, the marked anemia, the

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lesions of the skin, the enlargement of the spleen, and the presence of the diplococcus in the blood indicated clearly that we had to do with a case of infective or ulcerative endocarditis. The infection affected profoundly the cardiac muscle, while the valves escaped with less damage. The occurrence of embolism could not be demonstrated, nor did the severe chills and sweats occur that mark the disease in adult life. Treatment was of little avail, and mainly symptomatic. Arsenic, iron, and quinin were given for long periods, but did not have any specific influence in cutting short the disease, although they seemed to mitigate its severity. Theocin proved of great service as a rapid and powerful diuretic. Three to five cc. of 4 per cent. solution of collargol were injected into the veins of the arm on alternate days for twelve days, but failed to produce marked results. The leukocytosis increased decidedly (from 31,400 to 37,600) after the first injection. The temperature curve was not appreciably influenced by collargol, although the degree of daily variation was less after the first and fourth injections.

At the present time, six months after leaving the hospital, the boy's heart shows traces of his infection in the shape of an indistinct prolonged first sound, followed by a very soft systolic murmur conveyed faintly into the axilla and an accentuated pulmonary second sound, but there is no distinct evidence of hypertrophy or dilatation. The patient has gone back to his usual mode of life, apparently as well as ever, and has no subjective symptoms of heart disease.

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A CASE OF INTERLOBAR EMPYEMA FOLLOWING PNEUMONIA.

BY JOHN H. JOPSON, M.D.,

AND

JAMES E. TALLEY, M.D.,

Philadelphia.

MEDICAL HISTORY BY DR. TALLEY.

O. S., age 13, female, colored, was admitted to Dr. Musser's service at the Presbyterian Hospital, February 2, 1905. The family history was negative. Previously she had had measles, mumps and whooping cough. The present illness began four days before with a chill in the afternoon, followed in two hours by a second. She complained of pain in the epigastrium, at the base of the right side of chest posteriorly and for the first four hours also in the legs. Considerable cough and rusty expectoration began the day after the chills. On admission the area internal to the left scapula showed harsh breathing and a few crepitant râles, but on percussion this area was more resonant than the right side, and the roughened breathing and crepitant râles were probably due to an early and transient congestion, as there was no further development of consolidation at this point. On the right side anteriorly from the apex to the second rib there was, on percussion, a slight dulness, and to auscultation the breath sounds were harsh. The right base, the first site of severe pain, gave characteristic signs of consolidation a day later, the process having probably begun at the centre and worked toward the surface. Three days after admission the consolidation had reached its maximum; posteriorly, it extended from liver dulness to the apex, and anteriorly, from the apex to the third rib. The crepitatio redux first appeared in the right base on the ninth day, but was delayed in the upper lobe until the twelfth day. This was probably the true crisis, as the respirations, which had been running from 48 to 72, with an average of about 60, fell to 32 and later to the twenties; the pulse, which had been running at about 120, fell to 104, but went up again, and until the operation ran from about 108 to 116. The temperature, which had been running between 103° and 104°, fell only to 102°, and persisted between 101° and 102°. At first we thought the persistence of fever was due to otitis, the patient having complained of pain in both ears on the ninth day, and on

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the tenth day the left ear showed a purulent discharge. The one culture made from the discharge was negative. However, the remittent temperature persisting day after day, even when the otitis showed a tendency to improve, made one think of other causes for its persistence.

The leukocytosis kept in general about the same level throughout. It was 28,800 soon after admission; on the eighth day it was 45,200, the highest count, and this was at the time the otitis began. Nine counts from this time until pus was actually demonstrated in the chest gave an average count of 32,066. Three examinations of the sputum on the twelfth, fifteenth and nineteenth days, showed pneumonococci, streptococci, leukocytes, but no tubercle bacilli.

The possibility of an endocarditis was considered, as on admission there was only a very marked systolic murmur, heard best at the pulmonary area. This decided murmur persisted throughout, became more pronounced later, and was supposed to be due to some enforced deviation of the pulmonary artery by the inflammatory process, as the second pulmonary sound was clear, though much accentuated. Later, a well-marked mitral systolic murmur developed, with good transmission, and the apex beat was pushed farther to the left. However, this was probably due to a slight dilatation of the heart, and the murmur was one of relative insufficiency.

The hectic-like temperature, the development of abundant indicanuria, and the whole aspect of the patient, made pus somewhere probable, though the classical sweats and chills were absent.

Repeated examinations of the chest gave no signs of general effusion. The fluoroscope and skiagraph were not used at this time, largely because the patient was in the pavilion apart from the main building, where the x-ray room is situated. Again, the wide-spread consolidation of the pneumonia made the x-ray of doubtful utility in locating any circumscribed collection or collections of pus. Following Musser's rule that "in suspected loculated empyema or effusions the point of puncture should be at the side of greatest dulness and least fremitus," an exploratory aspiration in the seventh interspace, anterior axillary line, was made on the nineteenth day, and only a few drops of clear serum were obtained after penetrating the gristle-like pleura and moving the needle in various directions. A few days later a second aspiration was tried in the eighth interspace, posterior axillary line, and though

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the needle penetrated its full length and was moved in various directions, only a little serum, lymph and blood were obtained. Some days later, the conditions remaining the same, Dr. Jopson aspirated the chest in the posterior axillary line opposite the spine of the scapula through the sixth interspace, and after some manipulation of the needle, succeeded in obtaining pus at a depth of about two inches. Dr. Jopson will describe the subsequent operation and the result.

That the process was a loculated empyema and not a pulmonary abscess is best proven by the character of the pus obtained. It showed no pulmonary nor elastic tissue and no cholesterine nor haemotoidine crystals, all or some of which should, according to Osler, Musser, Leube, Strumpel, Eichhorst and others, be obtained in the pus of pulmonary abscess. That it was an interlobar empyema, situated between the upper and lower lobes, appears most likely from our later fluoroscopic and skiagraphic study of the case with Dr. Newcomet. With the fluoroscope even two weeks ago we could still distinctly see the shadow of a consolidation in the right chest. With crosses of lead strips as markers, and careful centering of the anode, we demonstrated that the shadow of the consolidation lay in a line drawn from the junction of the fifth and sixth thoracic vertebra posteriorly to the sixth interspace anteriorly.

A more recent skiagraph, made in as few seconds as possible, with the chest held at rest in full inspiration, shows the shadow on the right corresponding to the fluoroscopic image. Also as we had noted on the screen, a dark area at the right base shows that the pleura is thickened and perhaps resolution incomplete. The by far less marked shadow on the left we could not understand until we recalled from the history of the case, that this area was the very first place where crepitant râles and bronchial breathing were heard, but the process here subsided without running on to full consolidation. It is not unlikely that these less marked shadows are in part at least due to thickening of the pleura. However, the shadow to the right is distinct, and both screen and skiagraph show that it lies in general in the area of the commissure, between the upper and lower lobes.

SURGICAL NOTE BY DR. JOPSON.

The patient was transferred to the surgical ward on the 4th of March, the day following that on which we had succeeded in aspirating the encysted collection. Operation the same day. The

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diagnosis was empyema, and no suspicion of an interlobular collection was entertained. The patient's condition was poor; she was exhausted by her long illness, was emaciated, and looked septic. She took ether well. Aspiration in the seventh interspace, posterior axillary line, yielded pus. The incision opened the pleura at this point, the centre of the incision corresponding to the posterior axillary line. There were universal light adhesions binding together the parietal and visceral layers of the pleura, and obliterating its cavity, but no fluid was found, and after separating adhesions for an area of about six inches around the wound no collection was encountered. As we were close to the dome of the diaphragm at this point, and as pus had been withdrawn by deep puncture before incision, we concluded that the diagnosis now lay between an abscess of the lung and a subphrenic collection pushing up the diaphragm, at a deeper level. The aspirating needle was introduced through the lung and pus again found in an upward and backward direction. Two inches of the seventh rib were resected, the lung stitched to the chest wall around the edges of the wound, the needle again introduced into the abscess cavity, followed successively by a grooved director, hemostatic forceps, and the finger, and a large cavity discovered in the posterior portion of the thorax, bounded in front and to the outer side by lung tissue, and posteriorly approaching near the chest wall. Whether any compressed lung tissue intervened between the cavity and the parietal pleura at this point could not be determined. The pleural cavity, anterior to the incision, was packed off with gauze strips, and two rubber drainage tubes of good calibre were inserted into the abscess cavity. After operation the patient reacted nicely under free stimulation. The abscess cavity drained freely. The temperature continued moderately elevated for about six days, when it began to touch normal in the mornings. There was some evening rise, and occasionally slight elevations for several weeks longer, the wound continuing to discharge in decreasing quantity. The tubes were kept in situ for several weeks, being finally removed on April 12th, when the discharge was slight in amount, and thereafter the cavity was drained by a gauze strip. At this time (May) there is a small sinus several inches in length still present, which will probably be some weeks longer in closing. The patient has gained at least twenty pounds in weight, the temperature has been normal for a month, and she seems to be in perfect health.

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NOTE ON A SERIES OF CASES OF STOMATITIS.

BY HORACE H. JENKS, M.D.,

Philadelphia.

The purpose of this note is to call attention to a variety of stomatitis of which 12 cases have been seen this spring at the Children's Hospital, of Philadelphia, in the services of Dr. Griffith and Dr. Hand, through whose kindness I am enabled to make this report. The first 6 of these cases occurred within a few days of one another, and the rest have appeared at intervals of a few weeks.

CASES OF STOMATITIS.

Name.	Hospital Number.	Date of Admission.	Disease.
1. Lillian C.	124	3-15-05	Typhoid fever.
2. Joe S.	128	3-17-05	"
3. Harry D.	131	3-18-05	Pneumonia.
4. Mary K.	135	3-20-05	"
5. Pearl H.	138	3-21-05	"
6. Anna S.	142	3-23-05	"
7. Israel E.	147	3-26-05	"
8. Louis F.	152	4- 5-05	"
9. Thelma M.	179	4-22-05	"
10. Albert W.	200	5- 6-05	Typhoid fever.
11. Cora B.	207	5-10-05	"
12. Rosina d'O	226	5-21-05	"

The first case was seen on March 24, 1905, in a little girl, Lillian C., white, age three years. She was suffering with a mild attack of typhoid fever. Five days after her admission (the twelfth day of illness) a decided flushing of the right cheek below the malar eminence was noticed, and examination of the mouth showed an ulcerated patch about the size of a dime, opposite the upper anterior molar tooth on the mucous membrane of the right cheek. There was slight induration of the tissue surrounding the ulcer. No marked fetor of the breath was observed. The edges of the ulcer were well defined, but slightly irregular, and raised above the surrounding mucous membrane which was reddened and inflamed. The surface of the ulcer was covered with greenish black necrotic material, part of which could be scraped away, but not all. Cultures and cover-glass preparations were made from the ulcer. The cover-slip preparations showed at once many short polar-staining bacilli, resembling the Klebs-

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Löffler bacillus. The cultures the following day showed similar bacilli, and the culture sent to the Board of Health was reported as positive for the Klebs-Löffler bacilli. Fearing the possibility of noma the child was at once operated upon by Dr. Hodge, the ulcer curetted, and its base and edges touched with pure nitric acid. The child was placed in the isolation ward and 4,000 units of diphtheria antitoxin administered. The next day a small ulcer had developed upon the left cheek, at a corresponding point to the one on the right, and another below the lower incisor teeth, between the gum and lip. This was the only spot at which the gum was involved. The submaxillary lymph nodes on the right side were swollen and evidently tender. The child was very fretful and took her milk poorly. Salivary secretion was increased. Cultures from the ulcers were reported as positive for Klebs-Löffler bacilli for fourteen days. In eighteen days from the onset of the stomatitis the ulcers were entirely healed, and the child discharged. This was the only case with visible external involvement of the cheek, and the only one which resembled at all a gangrenous stomatitis.

The second case was noticed the day after the first was seen. It occurred in another ward, in a boy, Joe S., age three years, white, in the second week of a moderately severe typhoid fever. His ulcers also developed one upon each cheek opposite the anterior molar tooth, and one on the mucous membrane of the lower lip. They were more superficial than in the previous case. The gums were pale and not bleeding. These ulcers spread slightly, that on the right cheek extending almost to the angle of the mouth from opposite the anterior molar tooth. Cultures from these ulcers were reported from the Board of Health as showing Klebs-Löffler bacilli. The child was removed to the isolation ward where he remained for twenty-two days before cultures from the ulcers were returned as negative for Klebs-Löffler bacilli.

The third case was noticed three days later in the Baby Ward, on another floor of the hospital. This case was seen just as it was beginning. There was a small yellowish-white ulcer on the right cheek, opposite the anterior molar tooth, a larger one about one-half inch in width on the mucous membrane of the lower lip, and one the size of a small pin head on the left cheek. There was no noticeable glandular involvement in this case, but fetor of the breath was marked. Later three small, pin-head sized ulcers de-

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veloped on the lower lip. One of these grew larger, extending into the fold between the lips and gum and up the gum to the lower incisor teeth. These ulcers were healed in eleven days.

The remaining 9 cases were very similar to one another in appearance and course.

The age of the children varied from eleven months to three years; most of the cases were in children between two and three years of age. In none was there evidence of scorbutus. Two of the cases occurred in children who were seen to be biting their lips frequently, before any ulcer was seen. Five of the cases occurred in children suffering from typhoid fever, and seven in those ill with pneumonia.

The lesions were multiple in every case but one, when only a circular ulcer about one-fourth inch in diameter was seen, at the junction of the lip and gum below the incisor teeth. In the other cases the lesions numbered from three to five, usually three, and in practically the same situation in each case. The disease did not originate on the gum in any case, as far as could be determined, and while the gum was affected when first seen, in one case (Joe S.) the ulcers appeared further advanced on the cheek and lip than on the gum itself.

In 10 of the cases the appearance of the mouth was practically the same. In some cases the ulcer would appear first on one cheek and then on the fold between the lip and gum, below the incisor teeth, or *vice versa.* But by the second and third day of the disease the appearance of the mouth was as follows:

On the mucous membrane of each cheek, opposite the molar teeth at about the level of the meeting-place of the upper and lower jaw, is an ulcer, usually fairly circular, in size about one-fourth inch in diameter, but varying from that of a head of a pin, to one extending from behind the molar teeth to the angle of the mouth, about one and one-quarter inches (this in the later stages). A similar ulcer is present at the fold between the teeth and gums below the incisor teeth. The color is a yellowish-white—in the earliest stage of the disease—being surrounded in one case by a distinct pink areola. There is at first no marked excavation. The edges of the ulcer are clearly defined, slightly irregular, and not elevated. The surrounding mucous membrane is either unchanged, or slightly reddened. Breath is not offensive, salivary secretion slightly but not markedly increased. Pain is not evidenced, except when the ulcer is touched, when it appears to be

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tender. The ulcer tends to spread quite rapidly along the mucous membrane, and but little as to depth. In the later stages there is on each cheek and on the lower lip a yellowish-white ulcer, its floor composed of small granulations, its edges red, distinct, slightly elevated above the surrounding mucous membrane, which is now reddened, slightly indurated, with, in some cases, quite marked injection of the capillaries. This continues for from six to twenty days, the yellowish-white color remaining, the ulcer gradually becoming smaller, with no new ulcers forming in any case after the second or third day. In the more severe cases the submaxillary lymph glands about the angle of the jaw are swollen hard and evidently tender, but showing no tendency to suppurate. The tongue is not noticeably swollen in any case. It is coated or not, apparently according to the condition of the digestive system.

Other than the appearance of the ulcer the symptoms were not marked, as the children were all sick with other diseases. There were no such symptoms as fever, prostration, vomiting, diarrhea, or anorexia that could be ascribed to the condition of the mouth alone.

The principal interest of the ulcer was in its diagnosis from ulcers of the mouth, usually described as ulcerative or herpetic (aphthous) stomatitis. The differences between such diseases of the mouth and that seen this spring at the Children's Hospital, are, as far as can be ascertained, as follows:

From ulcerative stomatitis this form differs in the absence of offensive breath and swollen, spongy gums. The tongue is not swollen in the latter form. Here, too, the gums rarely bleed, as they frequently do in the ulcerative form. Salivation is not as marked as in the ulcerative form. An important differential point is that in this latter form of stomatitis the ulcerative process begins on the mucous membrane of the cheek or lip, whereas in the ulcerative form it is apt to begin at the junction of the teeth and gums. A dirty yellow deposit at the junction of the teeth and gums is more frequently observed in the ulcerative form than in that seen at the hospital, but upon removal of this deposit bleeding does not occur in the latter form as it frequently does in the former. There is evidently more pain in the ulcerative form, from which also recovery is as a rule more rapid. There are also many points of similarity, such as will suggest themselves, between this hospital form of stomatitis and the true stomatitis ulcerosa.

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Between this epidemic form and the herpetic form of stomatitis there are also many differences and many points in common. In the herpetic or aphthous form there are usually many lesions present, in the hospital form not over four or five. Another important point is that in the herpetic form larger ulcers are formed by coalescence of smaller ones, whereas in the hospital variety they are formed by extension from the original focus, these latter also extending deeply, as they never, or at most very rarely, do in the herpetic form. The lesions in the herpetic form are vesicular, whereas in the hospital form there is distinct *loss of tissue.* These latter ulcers are more yellowish than are the herpetic vesicles. The lesions in the hospital form were present only on the inner surface of the cheek, lips, or on the gum, just below the lower incisor teeth. No lesions were observed on the tongue or palate (except in case number 11, Rosina d'O., hospital number 226, where, in addition to yellowish-white ulcers on the lower lip, and one small ulcer on the left cheek, there were two ulcers on the soft palate, and a long, narrow, very superficial ulcer on the uvula).

It is to be regretted that we did not have at the hospital facilities for the complete bacteriological study of these cases. The supposition that Klebs-Löffler bacilli were present in cases 1 and 2 (Lillian C. and Joe S.) was based merely on the report from the City Board of Health, and on the study at the hospital of cover slip preparations made from culture tubes of Löffler's blood serum after from twelve to twenty-four hours' growth.

In conclusion it should be said that this note is not written with the intention of describing a *new* form of stomatitis, but merely to show how one variety of stomatitis will merge into another, and that while it is easy to make a diagnosis of stomatitis it is at times most difficult to designate accurately the variety that is present. The disease at the Children's Hospital most closely resembled the aphthous form, but it differed from it in being more severe as regards destruction of tissue, less painful, with the ulcers fewer in number, spreading by extension, and not by coalescence of smaller ones. It differed from the ulcerative form in the (usual) absence of offensive breath, profuse salivation, swollen, spongy gums, and by the ulcerative process beginning on the cheek or lip, and by its slow course.

It has been on account of these differences that the cases seemed to be of sufficient interest to report to-night.

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